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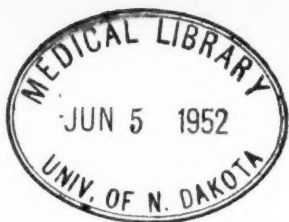
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CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

I. ANGINA PECTORIS SECONDARY TO ABDOMINAL ADHESIONS

THE patient is a married woman, fifty-five years of age. She was first seen on November 15, 1923, at which time she gave a very extensive history.

In 1894, when she was twenty-four years of age, the patient had a resection of the right rib for a bony tumor. Three years later, when the patient was twenty-seven years of age, the ovaries were removed, and she states that a stricture was removed from the rectum. We have no knowledge of what the last one of these operations could have been, but the former resulted in an artificial menopause. Three years later she was given gas for the removal of her teeth. A hemorrhage resulted, which kept her in bed for nine days. Five weeks after the teeth were removed she developed pain in the right axilla, and states that some enlarged glands found there, which were removed, were tuberculous. She also declares that an infection followed the removal of these tuberculous glands, which lasted six weeks. Since that time her right hand has always been swollen to some extent. Associated with the swelling of the right hand there has been a feeling of pressure in the right side of the neck and arm, and this feeling has gradually worked into the right hip. From this she

The next number of the MEDICAL CLINICS (May Number) will also be devoted to Chicago and will contain the Clinics from that city not reported in this number.

states that she was laid up about six months. Following this she had acute appendicitis. The appendix was removed and also a complete hysterectomy was done. The only comment which a medical man can make in regard to all these operations is that undoubtedly many of them were unnecessary, and that the surgeons of her early life must have been very industrious.

Previous to all of her troubles she had been married and gave birth to a son, who is now thirty-two years of age. She says she has never been well since his birth. She was never pregnant except upon the one occasion. Her husband was a man of good health and habits, a druggist by occupation. I saw him in consultation previous to his death from diabetes. He had never had any venereal history and a Wassermann test done upon the patient on November 16, 1923 was negative.

The patient's complaints when I first saw her November 15, 1923, was of a terrible aching in her legs, with a sensation of deep swellings in them. She was somewhat stiff in the joints of both lower extremities. In addition to this localized disturbance she complained of a certain amount of aching all over her body. This grew worse from September, 1923. Cross-examination disclosed the fact that four years previous she had had a periosteitis of the left tibia following an injury. This periosteitis had kept her in the hospital five and a half months, and was cured by Dr. A. J. Ochsner. The pains in the leg bothered her at night, but they were no worse then than at other times. When her arms were in one position any length of time her hands got numb, but there was no numbness in her feet, merely a peculiar feeling in the heels and occasionally a prickling sensation in the toes. The pains which she had were deep seated like a bone ache and not darting. She had nocturia at 5 A. M., but otherwise no renal symptomatology. Urinalysis is normal.

Her weight went down to 95 pounds four years ago when she had the periosteitis. Since then it has gradually increased, and in this year since March her weight has risen 20 pounds, so that she now weighs 137. Her teeth have been removed. Her tonsils were removed a year ago. The gall-bladder shows no pathology. She has no pelvic disorder. Her blood-pressure is

156 systolic and 90 diastolic. The heart and lungs are normal. She recovered from the pains rather rapidly by the use of the saturated solution of potassium iodid in 10-minim doses.

After being cured of these pains she was not seen again until November 13, 1924, a year less two days later. At this time she complained of gas in her stomach and bowel. The onset of this disturbance came two days after she had broken her ankle, for which she had gone to the hospital. She had not lost any weight, nor had she vomited. She stated that the gas took her breath from her; in her words, "it hit her heart and she would gasp for breath." She complained of belching and soreness beneath the right rib. Since September 12th she has lost 8 pounds. She had a bad taste in her mouth and declared that fumes came up when she awakened after a nap in the daytime. Shortly after November she went to Biloxi, Miss., to spend the winter, and I did not see her again until May 8, 1925.

She came back looking very pale, saying that she had been very poorly and that she had been under a physician's care. She complained of being very dizzy. At Biloxi she got a good deal of benefit from citrocarbonate, but before she left for Chicago she acquired a terrible pain in the epigastrium. She says she gets terrible pains in it, which come on after eating and may last three hours. She states that the pain which is chiefly in the upper midthorax does not radiate into the left arm, but goes straight through to the back. While she was sitting in a chair about 3 o'clock in the afternoon she was suddenly seized with a spell similar to the one she was trying to describe. It began with evidently terrific pain in the chest. Her pulse was found to be 108, was very unequal, and arhythmic. On listening with the stethoscope there was evidence of what used to be called delirium cordis. The attack was very evidently one of angina pectoris, and proved so severe that it was necessary to keep her in the office for a period of three hours and to send her home with a nurse in a taxicab. During all the afternoon the heart was very arhythmic and unequal at times. The pain was extremely severe and was not very well controlled by either nitroglycerin or morphin sulphate. During the height of the

attack the aortic arch showed no murmurs. She was of the opin-
ion that exercise did not bring on an attack, and said that she
never knew when it was going to come. She did state, however,
that it might come in the night after retiring, which is very
characteristic of true angina pectoris. She states that she has a
subjective feeling of a twisting of the heart. She does not admit
that the attack scares her, but she says it tires her completely
out; in other words, she has no *angor animi;* she does have
symptoms of arrest. When the attack comes on she has to
stop and be absolutely inert. The best description is of a ter-
rible pressure in her chest, as though some one had his heel in
the chest and was digging in with it.

We were very glad that the patient did not die in the office.
We had arrangements made so that she might immediately go
to the hospital. The fluoroscopic examination which I made
personally on May 11th showed the heart moderately dilated
laterally and the myocardium gave one the impression of aton-
icity. The aortic arch was just a little widened and did not pul-
sate, being apparently rigid. The pulsations in the left auricle
were easily visible and there was no trace of auricular fibrilla-
tion. The lungs appeared to be clear and the diaphragm pre-
sented normal contours. On entrance her blood-pressure was
130 systolic and 80 diastolic, as against 156 systolic and 90
diastolic in 1923. This fall in blood-pressure seemed somewhat
ominous and confirmed one in the opinion derived from the
fluoroscopic examination that the myocardium was not as effec-
tive as it had previously been. The rigidity of the aortic arch
seemed to indicate that the blood at times tried to distend it,
and being unable to do so because of the rigidity, occasioned
the tremendous attacks of angina pectoris. It seemed to con-
firm Sir Clifford Allbut's theory of the cause of angina pectoris
as being essentially aortic.

There was no doubt that the patient was suffering from
severe angina pectoris, associated with vagatonic symptoms.
The great question was, Why? Angina pectoris, in general, at
the age of fifty-five is apt to be luetic, but our knowledge con-
cerning the Wassermann test and her previous history together

with our knowledge of the husband made it seem very improbable that there was any luetic element in her case. We were therefore of the opinion that we would have to seek another cause for her angina. Angina pectoris may also be infectious from other sources than the *Spirochæta pallida*, but no other evidence of infection could be found in her body. Gradually with rest the angina disappeared, so that in the course of a month or two she was very comfortable, but with her increase in comfort from the improving angina a symptom began to develop which disclosed to us the apparent cause of her attacks.

After she had been in the hospital for perhaps a month she again began having severe abdominal pain. The attacks of pain occurred almost invariably shortly after eating, and would last a variable length of time, usually a matter of several hours. The nights became very troublesome. She would have her supper and shortly thereafter pain would begin in the abdomen and last until 2 or 3 o'clock in the morning. The pain was evidently due to a contraction of the unstriped muscle-fibers. It was located in two chief places, the right iliac fossa and deep in the pelvis. This pain was relieved to a slight extent by the application of heat and by pressure. There was, however, some rigidity of the abdominal wall and also a slight amount of tenderness, as though the peritoneum were affected as well as the unstriped muscle-fiber of the large bowel. The patient left the hospital on June 20th fairly free from her angina, but with quite a good deal of abdominal pain and attempted to get on without further medical aid.

On August 2d, however, the pain became so intense that she again entered the hospital, where I saw her on the 3d. We felt that there must necessarily be, because of the operation of hysterectomy and appendectomy and the removal of both ovaries, some adhesions responsible for the pain. We therefore gave her a barium enema and made two plates, one of which is here reproduced (Fig 216). The interpretation of the plate proved to be very simple. It accounts at once for the two locations of the pain. The pain in the right iliac fossa was found to be due to kinking of the colon, which was looped upon itself in such

a way that it did not readily permit the passage of gas and fecal matter. The contention that she could feel gas stuck in the right side was apparently confirmed by the x-ray plate. The worst pain, however, was always deep within the pelvis. There being no pelvic organs remaining, it was evident that the dis·
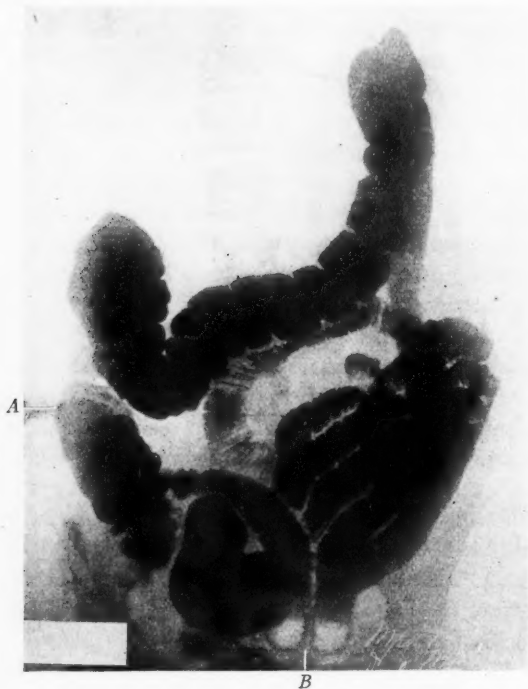
Fig. 216.—Case I. Note cross-bands of adhesions, arrow *A* binding two sections of the ascending colon together; and arrow *B* showing two parallel loops of small bowel in the pelvis, these loops being filled with air and connecting posteriorly. The latter observation shows in the original plate, but not in the reproduction.

turbance must be adhesions of some sort, and the plate shows very nicely a loop of bowel going down into the pelvis and being kinked upon itself, containing gas at its bend. The x-ray plate shows this descending loop of the small bowel with the distention of the gas very beautifully.

Between May 8th and June 20th, when she left the hospital, she had had very few attacks of anginal pain. At first the attacks of angina occurred at intervals of two or three days, but by and by they died out both in severity and frequency. In August, however, the attacks of angina began again to assert themselves to some extent, so that the patient was able, as she thought, to make out a connection between the abdominal and thoracic pain. This was evident also to her physician. It occurred to me that hers was probably one of those instances of the sympathetic nervous system origin of attacks of angina pectoris.

Here one must make a digression. Angina pectoris is undoubtedly organic, but at times is probably functional. We used to divide angina pectoris into the true and the false, but such a division is wholly artificial. All angina is angina. There is no such thing as true and false angina. Angina is, as a rule, organic, and yet a few cases of reflex sympathetic nervous system angina undoubtedly occur. It seems as though in certain conditions of the gall-bladder and the abdomen that reflex disturbances may take place which lead to anginal symptoms of the aortic arch or bring on attacks of angina pectoris. We began to wonder whether it might not be so in this case, and so we began to get rather eager for an operation. We explained to her that these adhesions of which we were so sure could probably be causing the attacks from which she had so severely suffered. She was at length so persuaded of our contention and also by the severity of the pain in the abdomen that she finally decided, with her son, to have the operation performed.

The operation was done by Dr. E. Wyllys Andrews. He was not wholly persuaded to my view of the case because he labeled his operation "removal of adhesions and exploration." We were of the opinion that there was not very much left to explore, and that the pain, being localized to the points of pathology in the x-ray plate, was completely and wholly accounted for. Gas and ether were used as the anesthetic. A midline incision was made at the site of the former incision and the scar tissue removed. An adhesion was found in the right

colon, the loop of the colon being fastened upon itself just above the cecum exactly as in the x-ray plate. This adhesion was broken up and covered with peritoneum. The entire abdomen was then explored and the other adhesive area was found to be exactly as in the x-ray plate. A loop of small bowel had herniated itself down through some adhesions of the small intestine and had become adherent in the pelvis. This loop was pulled out of its malposition, the adhesions being broken up and the peritoneum covered in as well as could be. In addition, a band of adhesions was found crosswise in the abdomen fastening the transverse colon to the anterior abdominal wall. This band, which did not show in the x-ray plate, was cut and prevented from doing any further damage. The abdomen was then closed without drainage.

Following the operation the patient did reasonably well. Her hemoglobin, however, which previous to the operation had been 87 per cent. fell to 48 per cent., and the red cells from 4,910,000 to 3,250,000. There was nothing to account for her gradual anemia, though it was somewhat disconcerting because of the fact that a fractional test-meal showed no free hydrochloric acid. The white cells were 9200, with 68 per cent. polymorphonuclears and 32 per cent. small lymphocytes, so that there was no evidence of any untoward condition. With appropriate treatment the blood rapidly improved, so that whereas on September 21st the hemoglobin was 48 per cent., on October 1st it was 70 per cent., and the reds had risen in the meantime to 4,030,000. By October 3d the hemoglobin was 75 per cent., the red cells 4,080,000, and the white cells 7700. By October 27th the patient was so far recovered that she was dismissed cured. She has had no attack of angina pectoris since the operation, and we believe that she will never have another attack unless she gets it from an entirely new cause.

It is perhaps too soon to say that her case proves the existence of a reflex angina pectoris, but to me it seems to prove it. A patient who had so severe an attack as this patient had in my office, where I feared she would die, does not usually recover completely if this be due to organic disturbance. The

rigidity of the aortic arch at the time of the fluoroscopic examination in May may well have been due to anginal symptoms at that time.   Our old conception of angina pectoris as being at times due to angiospasm is apparently confirmed by this case. We must admit, I think, in the future that if a woman, fifty-five years of age, without proper etiology for attacks of angina pectoris in the shape of lues or other infections, has such attacks of angina these attacks may be due to some reflex irritation of the sympathetic nervous system.   It is not a far-fetched idea that a severe spastic contraction of the colon and small bowel may lead to marked perturbations of the afferent impulses from the sympathetic nerve centers.   It is not difficult to see how disturbances in the intestinal pneumogastric area can lead to reflex cardiac responses in the thoracic pneumogastric territory, so that explanation here of the relationship in this case between the abdominal disease and the angina can hardly be denied.
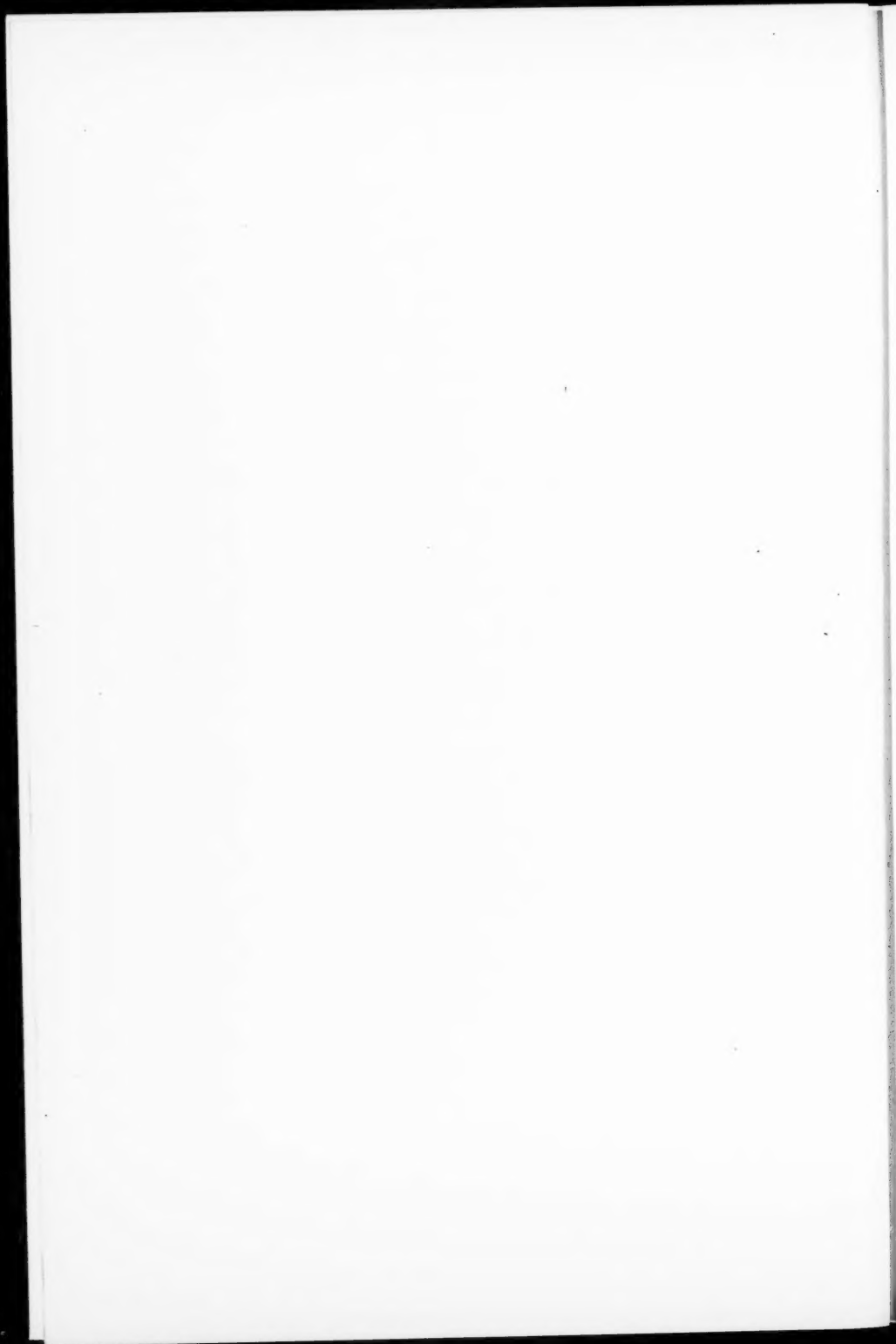
II. AN EXOPHTHALMIC GOITER OF PECULIAR TYPE

THE patient is a woman sixty-seven years of age. Her trouble began, as she thinks, somewhere about February 18, 1925. She says it began with an attack of fainting and that she fainted several times in succession. With these attacks of syncope, which were peculiar because they came on rather abruptly, she had smothering spells. After recovering from the faint she always vomited and felt very oppressed in the head. Before this she has always been well. Now she is extremely short-winded, even on talking, and at times she cannot even go upstairs. She has no swelling of the feet or ankles and she is at a loss to account for her condition. She weighed when she was taken ill 215 pounds. She has lost weight quite rapidly, so that now (in September) she weighs 132 pounds. She thinks that she lost the bulk of these 83 pounds in about four months' time. Her eyes are now rather protruding, so that she has an exophthalmos so marked that anybody can appreciate it at a glance. She has, however, a pulse which is extremely peculiar. One would think that it ought to be ranging from 140 to 150. Instead of that it is 78, rhythmic and equal. She has a marked tremor. Her face is flushed, being a kind of cyanotic pink, and the flush extends down over the chest. She states that she has always had such a flush, but her relatives say she is mistaken. Her chief complaint is terrible restlessness and nervousness. She feels driven every moment of her existence. She cannot sit still a moment. She suffers far more from the restlessness and nervousness than from any single thing. She is not apparently very much impressed by the loss of weight. She says she feels as light as a feather and yet she cannot go upstairs without being extremely winded. She was sent into the hospital at once and her basal metabolism found to be 97 +:

She was immediately put upon Lugol's solution in doses of 8 drops three times a day, and kept on it until she left the hos-

pital, October 31st, weighing 142¼ pounds, with the exophthalmos completely gone and with her pulse still 78, rhythmic and equal, entirely unchanged. Her strength, however, had returned. The nervousness and restlessness had completely disappeared and she regards herself as entirely cured. She says she does not wish to weigh above 150 pounds and she has only 7¾ pounds to go to be satisfied.

This patient's case is unusual from two points of view: First, her pulse has never been rapid as it ordinarily is in the usual case of exophthalmic goiter, and, second, she has no appreciable goiter that can be felt. She must have had a hyperplastically enlarged thyroid, but on palpation it could not be outlined. There was nothing whatever to be seen of a goiter on inspection, and yet the signs of exophthalmic goiter were so unmistakable that no one could fail to note them. There must, then, be a type of exophthalmic goiter without tachycardia and with emaciation, just as there are types of exophthalmic goiter with tachycardia and without emaciation.

This patient may serve as a text for a few remarks upon the thyroid gland. It is very difficult to classify thyroid cases pathologically and symptomatically because of the interlacing of the different types. In general, a good pathologic classification is into the simple hypertrophies and hyperplasias, the various kinds of adenomas and the exophthalmic hyperplasias. Symptomatically the cases may be best divided into the cases of hypothyroidism, corresponding to the first pathologic group, cases of hyperthyroidism, corresponding to the adenomatous group, and cases of dysthyroidism corresponding to the exophthalmic hyperplastic group. This patient belongs to the latter of these categories. Whereas iodin in the group of adenomas seems to make the patients very much worse, in the cases of dysthyroidism iodin in the form of Lugol's solution benefits the patients very much.

The thyroid gland undoubtedly is for the purpose of carrying on what may be termed our "iodin metabolism." Iodin is essential to the body and most individuals get it into their systems from the water which they drink. In certain localities in

our country the amount of iodin in the water is 1 part in 11,000,-000,000. This is true of the waters of the Great Lake region, so that iodin is extremely deficient in the locality of the Great Lakes. On the other hand, in the Mississippi Valley and in the Atlantic Coast District iodin is very plentiful in the water, so that the people of these districts scarcely have such maladies as exophthalmic goiter and simple hypertrophic hyperplasia. The diseases of the thyroid gland, therefore, are to a large extent dependent upon the distribution of iodin in the waters of the earth. In mountainous districts with granite formation, where large igneous rocks are very plentiful and iodin correspondingly rare, simple goiter and exophthalmic goiter are comparatively frequent. This is true especially in Switzerland and in the Himalayan Mountains, numerous cases existing both in Thibet and the Alpine districts.

The purpose of iodin in the body is undoubtedly to carry on the internal respiration of the tissues. This it does by promoting oxidation directly. Iodin is a great oxidizer and can burn up a good many useless tissues and substances in the body. The syrup of the iodid of iron is extremely valuable in getting rid of infectious granulomatous tissues in any place because of the oxidizing ability of the iodin which it contains. When iodin is insufficient in the body the internal respiration of the tissues suffers. As a result there is apt to be a certain amount of acidosis due to perversions of this internal respiration with rather inadequate scavengering of waste products.

Exophthalmic goiter must perhaps be looked upon as a composite disease. It seems not only to be due to a dysthyroidism, but there seems also to be associated with it a general intoxication from a peculiar form of by-products from imperfect tissue oxidation. The loss of weight in exophthalmic goiter and the excessive basal metabolism are not at all explainable on the basis of increased oxidation from iodin because in these conditions, so far as we know, iodin is actually deficient. The increased basal metabolism and intense intoxication of it are apparently due to by-products of a peculiar nature consequent upon some disturbance of its metabolism due to the iodin de-

ficiency. When iodin in the form of Lugol's solution is supplied to these individuals a better physiologic oxidation is promoted, disintegrating products cease to be formed, and the basal metabolism rate falls. Thus, in the case of the patient of whom we have just given the history, within three weeks' time the basal metabolism fell from +97 to +50. We do not know what it was on the day of her dismissal (October 31st), but it was undoubtedly less than 50, since she was gaining rapidly in weight, having gained $2\frac{1}{2}$ pounds during the last week of her stay in the hospital.

Our views concerning the fundamental nature of exophthalmic goiter must give place to some entirely new views. Whereas once upon a time we regarded it as due to an excess of iodin in the thyroid gland, we must now admit that it is not due to an excess, but rather to a deficiency; else how can we explain the relief that is obtained by giving iodin in the form of Lugol's solution to these patients? Years ago I noted, as many physicians have noted, that an occasional case of exophthalmic goiter alternated with myxedema. These cases used to be extremely hard to understand because apparently in earlier times they seemed to be alternations of excess of iodin metabolism with an insufficiency of iodin metabolism. From what we know at the present time it is easier to explain how the two conditions may be more or less coexisting simultaneously or consecutively. Myxedema is really a hypothyroidism, exophthalmic goiter is dysthyroidism with an iodin deficiency; both being iodin deficiency diseases it is easy to explain how they may at times be associated in the same individual.

The complexity of the problem of exophthalmic goiter is made greater by just such cases as the one reported. Here is a case of a woman who has lost 83 pounds, but who has never suffered from tachycardia, although she has shortness of breath and dyspnea. On the other hand, I am at present taking care of a young woman who during this last month has gained 10 pounds, after a loss of 58 pounds, and who has made a total gain of $41\frac{3}{4}$ pounds, but whose pulse ranges from 110 to 136 and who has a very marked exophthalmos. Both must be regarded

as cases of exophthalmic goiter, and yet they present pictures which are very different from one another, being alike in weight loss and different as regards pulse-rate. I have seen more than one instance of each of these two types of cases, and I can explain them only on the basis of a different sort of intoxication of the body growing out of the dysthyroidism of an exophthalmic goiter.

In both instances the sympathetic nervous system is undoubtedly very much irritated, and Müller's muscle by its contraction forces out the eyeball, giving rise to exophthalmos, but the two types of cases differ radically in weight, since in one case there has been a tremendous loss of weight, with but slight gain, and in another case both great loss and great gain. Moreover, the cases differ from one another in respect to the heart, the one now reported showing no tachycardia whatever and the other showing a very marked tachycardia. The cases are thus complicated by the fact that there may be great loss of weight with tachycardia, as well as a great loss of weight without tachycardia, and there may be cases of great gain in weight without corresponding reduction of the tachycardia.

There is only one conclusion which can harmonize these various types of exophthalmic goiter, that is, that the iodin metabolism is perverted in such a way by reason of the iodin deficiency that different kinds of toxic products are formed. We must assume that these toxic products are to some extent selective in action and that some of them lead to marked loss of weight, while others are exciting to the acceleratory fibers of the heart. These acceleratory fibers of the heart are probably sympathetic nerve-fibers running in the pneumogastric nerve, and the tachycardia is, therefore, to be looked upon in precisely the same way as the protrusion of the eyeball, namely, as a sympathetic nerve phenomenon. Nevertheless, the toxin may in some instances not affect the acceleratory fibers of the heart, as in the patient whose case has just been reported, though, in the main, they do so.

Undoubtedly there are cases of exophthalmic goiter without exophthalmos. I have surely seen instances of tachycardia, in-

creased basal metabolism tremor, nervousness, and all the usual symptomatology associated with exophthalmic goiter in individuals with hyperplastic goiter without adenoma. These are undoubtedly cases of the exophthalmic goiter type without exophthalmos. It would seem to me that in the course of time we are going to be able to differentiate between the various sorts of intoxication in dysthyroidism; to group the several sorts of cases of exophthalmic goiter. We shall have cases of exophthalmic goiter without exophthalmos; we shall have other groups without loss of weight; we shall have other groups without tachycardia, and presumably groups without tremor. At present the whole matter seems to be to some extent chaotic, and yet time will help us in the solution of the difficulty. Whether exophthalmic goiter is to be looked upon as a disease of the thyroid gland or whether there is an associated pathologic physiology of the adrenals, as seems to be indicated by the pigmentation of some cases of exophthalmic goiter, time and careful clinical observations alone will tell. The probabilities are that ultimately we shall look upon cases of exophthalmic goiter not as simple cases of dysthyroidism, but as complex cases affecting more than one ductless gland.

CLINIC OF DR. WALTER W. HAMBURGER

MICHAEL REESE HOSPITAL

DISEASE OF THE CORONARY VESSELS, ANGINA PECTORIS, AND "ACUTE INDIGESTION" (WITH SPECIAL REFERENCE TO THE CORONARY T-WAVE*).

FIVE years ago, in this clinic, I reported the case of a man dying suddenly following an acute sudden pain in the upper abdomen associated with temperature, leukocytosis and jaundice, and concluded that this man suffered from a sudden occlusion of his coronary circulation. Since that time "much water has passed under the bridge," and the entire subject of coronary disease, its symptomatology, pathology, diagnosis, and treatment has been amplified and clarified by the publication of many similar reports, some with electrocardiographic and autopsy findings. However, the clinical picture of coronary disease is still so new, and in many quarters unrecognized or unappreciated by the profession at large, particularly those atypical cases of coronary disease in which the outstanding symptoms are largely abdominal and in which the casual diagnosis is "acute indigestion," "ptomain poisoning," "gall-bladder disease," etc., that I thought you might be interested in the presentation of a number of other cases with some reference to the more recent literature, together with an attempt to more accurately describe and crystallize this interesting and important symptom-complex.

While many of these patients have the symptoms and findings of a cardiac or circulatory disorder, a considerable percentage of them present outspoken abdominal symptoms. Crohn[1] reports 6 cases in men, varying in age from forty-three

* With the technical assistance of Miss Helen G. Howard.

to sixty-one years, whose chief complaints were those of indigestion or flatulence, with expulsion of large amounts of gas by mouth and rectum, the belching of large amounts of air, with fulness of the abdomen, all of whom suffered with coronary disease. These patients insist that their symptoms are due to overeating and indigestion, accompanied by constipation. They complain of attacks of severe epigastric distress or cramplike pains after meals, not with the regularity of peptic ulcer, but irregularly and often shortly after an unusually heavy meal; not of constant recurrence or with characteristic pain, burning, and eructation, but vice-like, with relief by belching; in fact, in 3 of these cases a diagnosis of peptic ulcer had been made. The attacks of pain are often accompanied by an ashy gray facies, with beads of cold perspiration on the forehead, and with cold clammy fingers. Crohn speaks of the difficulty in convincing these patients as to the source of their trouble. Many of these patients in addition to the diagnosis of ulcer have been said to have gall-bladder disease and gall-stone colic, and it is, in fact, exceedingly difficult at times to differentiate these two conditions.

A case in point which I might mention briefly is that of C. L., age forty-four years, who on the night of October 19, 1920 at 11.30 o'clock, complained of severe, acute abdominal pain in the region of the umbilicus followed by an acute diarrhea. He attributed his attack to the eating of clam chowder. He had been under the care of a physician during the past three months for an unexplained albuminuria. Examination revealed a blood-pressure of 120/80, normal pulse and temperature, a white count of 17,400, 2 plus albumin in the urine, and a moderate enlargement of the heart both right and left, with a slightly accentuated second aortic tone. For a number of months he had been rising at night to pass water, with increased urination during the daytime. His electrocardiogram, taken ten days later, showed the findings of a left bundle branch block with the same findings six weeks later (Fig. 217). Electrocardiograms of this type, as we shall see later, are frequent in disease of the coronary vessels. In the five years which have elapsed since the

onset this patient has had no recurrence of his symptoms, although his electrocardiographic curve at the present time shows

NOVEMBER 1,1920. LEFT BUNDLE BRANCH BLOCK.

DECEMBER 15.1920. LEFT BUNDLE BRANCH BLOCK.

SEPTEMBER 30,1925. LEFT BUNDLE BRANCH BLOCK.
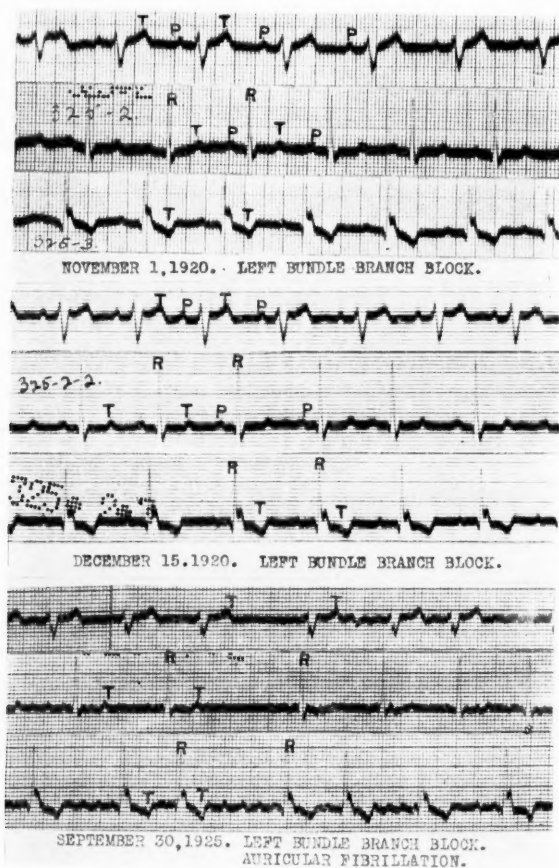AURICULAR FIBRILLATION.

Fig. 217.—C. L. "Ptomain poisoning." Acute abdominal pain and diarrhea following eating of clam chowder. Probable result of small coronary obstruction.

fibrillation of the auricle in addition to the involvement of the ventricle (Fig. 217).

While too much importance cannot be laid on the electro-
cardiographic findings, still if it were not for these very definite
evidences of bundle branch block in this patient, the true nature
of his attack might have been overlooked. As it is, I have no
hesitancy in saying that at the time of his so-called "ptomain
poisoning" he suffered from a slight obstruction of one of the
smaller branches of his left coronary artery.

An even more striking example is that of Mr. J. K., a coal
dealer, age forty-two years, who was referred to me by Dr. R.
P. Schuller, of Kokomo, Indiana, in October, 1923. This pa-
tient stated that four weeks prior to his entrance into the hos-
pital he suffered with a severe attack of "indigestion while play-
ing golf," so severe as to necessitate discontinuing his game and
to walk with assistance to the clubhouse. The attack of indi-
gestion consisted largely of severe epigastric pain in the region
of the xiphoid, rapidly passing toward the region of the heart
and to both shoulders and down to the abdomen. His general
health previously had been good, although the bowels had been
chronically constipated. He weighed 170 pounds. He had had
a slight trace of sugar in his urine for a year and a half and had
had a number of attacks of tonsillitis with fever. He was a
moderate user of tobacco. He had one boy, fourteen years of
age, and his wife was living and well and had had no miscarriages.

Examination revealed a heavy set, short, thick-necked in-
dividual, with rather florid countenance and some slight cyanosis
of chin and ears. His radial pulse was of good quality, but slightly
rapid, the left pulse distinctly smaller than the right. There
was some evidence of gum infection and some old discolored roots
in his lower jaw. The tonsils were enlarged and ragged, contain-
ing purulent material in the crypts. The left heart border was
one finger outside the nipple line, the right heart border at the
right sternal margin. The tones were faint and distant without
murmurs; the second aortic slightly accentuated. There was
slight tenderness over the sigmoid and colon. There was no
edema. Blood-pressure was 165/100. There was $\frac{1}{2}$ of 1 per
cent. sugar in the urine. White blood-count was 12,000, red
cells 5,470 000, hemoglobin 85 per cent.; differential count

showed 74 per cent. polymorphonuclears, 14 per cent. small mononuclears, 10 per cent. large mononuclears, 2 per cent. transitionals, and 1 per cent. eosinophils. Wassermann test was negative. Blood-sugar was 134 mg. per 100 cm., non-protein nitrogen 56. Complete gastro-intestinal x-ray examination resulted negatively except for the finding of a positive gall-bladder shadow. An electrocardiogram taken on admission showed the

OCTOBER 30,1923. MARKED INVERSION OF T WAVE IN LEADS II AND III. "CORONARY T WAVE"(PARDEE).

NOVEMBER 30,1923. INVERSION OF T WAVE IN LEADS II AND III STILL PRESENT BUT LESS MARKED.
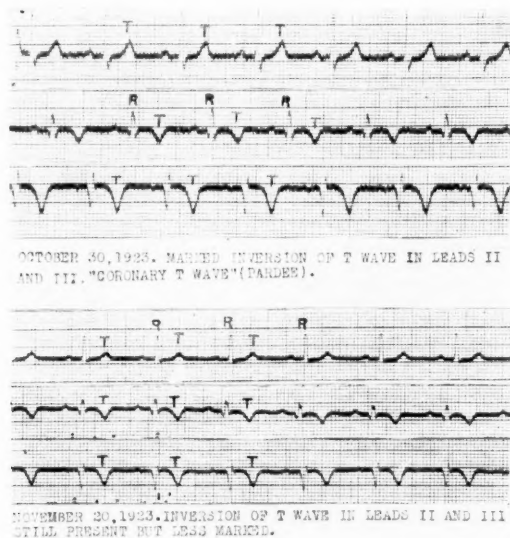
Fig. 218.—Mr. J. K., coal dealer, age forty-two. "Severe attack of indigestion while playing golf." Acute epigastric pain radiating to shoulder and precordium; probably result of coronary obstruction.

striking and characteristic inversion of the T-wave in the second and third leads (Fig. 218), which has been described by a number of writers as evidence of coronary vessel disease. Willius and Pardee[2] have particularly called attention to this so-called coronary T-wave in clinical cases, and Fred M. Smith[3] has produced the same experimentally in dogs by the ligation of the anterior descending branch of the left coronary vessel. These electrocardiographic findings in addition to the history and phys-

ical findings spoke for coronary vessel obstruction with result-
ing heart muscle degeneration, particularly in the region of the
apex of the left ventricle. In this patient we were able to repeat
his electrocardiogram from time to time, and it is interesting to
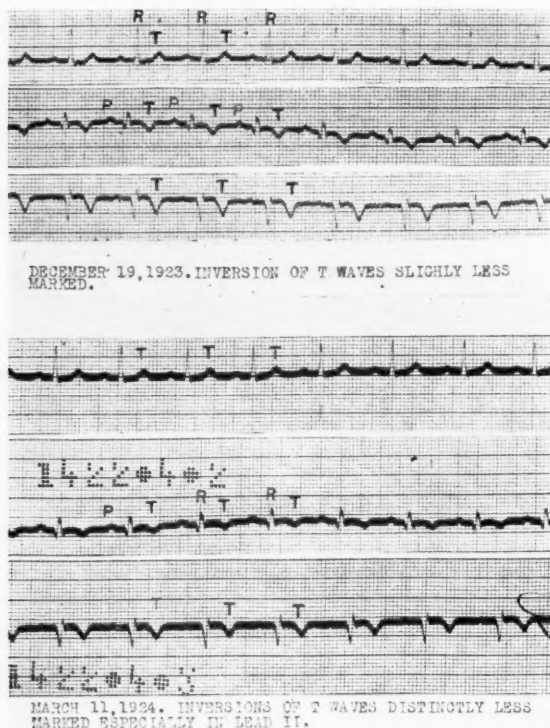
Fig. 219.—J. K. Gradual lessening of inversion of T-waves. Sudden death
on October 28, 1924, one year after original coronary accident.

see the rather constant and progressive disappearance of this
inversion (Fig. 219). The last curve (made in March, 1924)
showed an almost normal electrocardiogram.*

* Since this clinic was held a reply was received from the second follow-up
letter sent this patient to the effect that he had died suddenly October 28,
1924, seven months after the last electrocardiogram and just one year from the
onset of symptoms.

With the exception of these sudden attacks of so-called acute indigestion, these patients are remarkably free from any clear-cut and well-recognized group of symptoms; in fact, the indefiniteness, variability, and striking *lack* of uniformity of their symptoms is of itself suggestive, particularly in contrast with the more or less definite symptoms of actual abdominal conditions, namely, peptic ulcer, gall-bladder disease, disease of the pancreas, colon, etc. Nathanson[4] has recently commented on this same feature in a discussion of the clinical and pathologic features of disease of the coronary arteries, and speaks of the frequency of fulness, sense of pressure, pain in the upper abdomen, belching, nausea, and vomiting. He further discusses the similarity of these symptoms, with various gastro-intestinal disturbances, at times even simulating the picture of an acute abdominal lesion, and emphasizes the fact that the presence of coronary pathology undoubtedly explains some of the negative laparotomies and is the underlying cause of the many deaths of those dying of so-called acute indigestion, particularly in patients of advanced years. Willius[5] likewise recently has commented on the clinical features of coronary sclerosis and calls attention particularly to a group of so-called occult coronary sclerosis in which there are few if any symptoms referable to the heart and circulation.

ASSOCIATED CLINICAL FINDINGS

In addition to these symptoms arising apparently from the region of the abdomen there are a number of associated clinical findings which are not infrequently met with in these patients. These symptoms usually occur in men about forty or fifty years of age. These patients are frequently obese, corpulent individuals, with florid complexions, short, thick necks, with heavy arms and hands, with often evidence of atherosclerosis with increased systolic and diastolic pressures. They complain of slight dyspnea on exertion and often have belching and precordial pains on effort or following a moderately heavy meal. There is often some demonstrable enlargement of the heart to the left and not infrequently some difference in the size of the

two radial pulses. They not infrequently complain of distress
at night and of a cardiac arhythmia. They are often slightly
cyanotic about the tip of the chin, the lips, and the tops of the
ears. The association of these general findings with the symp-
toms of vague abdominal distress, particularly belching after a
heavy meal and attacks of ptomain poisoning and acute indi-
gestion, are of themselves almost sufficient to make a diagnosis
of coronary vessel disease. I recall in this connection the im-
portance of these general findings in the history of my first pa-
tient,[6] who died suddenly on the tenth day after arising from
bed. He had jaundice and leukocytosis in addition to the severe
abdominal pain, and the differentiation from a common duct
stone was made largely from the fact that some time previously
he had been under treatment by his physician for increasing
shortness of breath on exertion, a blood-pressure of 170/100,
and albuminuria.

Robey[7] has again recently emphasized the relationship and
differentiation between gall-bladder disease and coronary scle-
rosis and their occasional association in the same patient. The
difficulties of diagnosis are well represented in my second case
of five years ago, which, following operative findings, was proved
to be a case of so-called abdominal angina with chronic duodenal
ulcer simulating chronic gall-bladder disease.[6]

ANGINA PECTORIS

While many of these patients have few or no complaints and
some have only vague abdominal "indigestion," others have
more or less typical attacks of mild or severe anginal pain and
are diagnosed as angina pectoris. A case in point is the following:

Mr. M. S., age fifty-two, a carpenter by occupation, has had
heart trouble for three years and has been unable to work for
the past year because of the pain in his chest. The pain is des-
cribed as being in the upper epigastrium, has been coming on in
attacks three or four times a day, lasting ten or fifteen minutes,
often initiated by walking a short distance or climbing a few
stairs, sometimes quite severe, and characterized by a sense of
constriction, accompanied by dyspnea, but without radiation to

the shoulder or arm.   The pain is so severe as to at times make
it difficult for the patient to breathe.   He turns a grayish-green
color, with beads of perspiration on his forehead.   He had acute
nephritis when he was thirty years of age.   He has 5 children
living and well.   His father died of apoplexy.
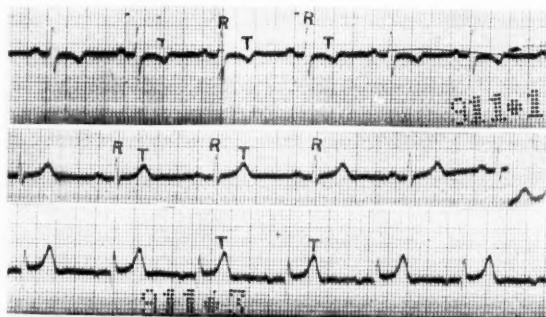
AUG.1922. - INVERSION OF T WAVE
IN LEAD I.INCREASED AMPLITUDE OF
T WAVE IN LEAD III.
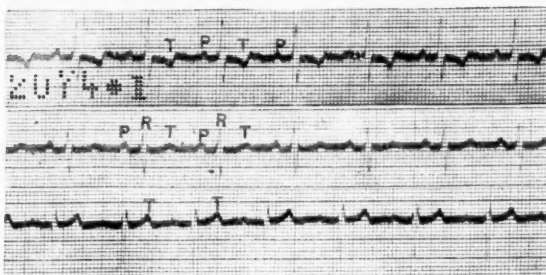
JUNE 1925. - INVERSION OF T WAVE
IN LEAD I. T WAVE IN LEADS II AND III
NORMAL . LEFT VENTRICULAR PREPONDERANCE.

Fig. 220.—Mr. M. S., carpenter, age fifty-two.   Typical angina pectoris.
Inversion of T-wave in Lead I, probably the result of disease of the coronary
vessels.   (Descending branch of left coronary artery.)

Examination (August 17, 1922) revealed a regular, slightly
rapid pulse, of good quality, fuller in the left wrist than in the
right.   His neck is short and thick.   The right heart border is
3 cm., the left 12 cm. from the midsternal line.   The heart tones

are faint, distant, and rapid. The liver is neither enlarged nor tender and there is no edema. The blood-pressure ranges from 100 to 120 systolic and 70 to 80 diastolic. The white count is 6600. The electrocardiogram shows again the typical inversion of the T-wave in the first lead which has persisted for a period of three years (Fig. 220). The last curve (June, 1925) shows substantially the same.

This man might then be said to have a quite typical angina pectoris, but I believe, in view of the inversion of the T-wave in Lead I, that we are justified in saying that the angina is on the
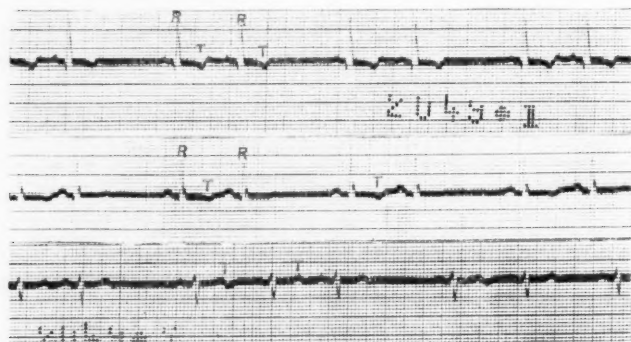
Fig. 221.—Mr. M. G., merchant, age seventy-two. Angina pectoris with generalized atherosclerosis and atherosclerotic aortitis. Recurrent auricular extrasystoles with bigeminy. Inversion of T-wave in Leads I and II. Coronary vessel sclerosis.

basis of a sclerosis and possibly thrombosis of the descending branch of the left coronary vessel, with some degree of myocardial degeneration in the region of the apex of the left ventricle.

Another example of this same type of angina is the case of Mr. M. G., age sixty-five years, a dealer in iron and coal, who twenty-seven years ago suffered with lead-poisoning. For the past fifteen years he has had a systolic blood-pressure of 200 mm. Hg. He complains at times of vague precordial pain, has had much belching, indigestion, and constipation. Examination reveals an oldish little man, somewhat shaky and cyanotic, with tortuous beaded vessels, without edema, enlargement of the liver

or dyspnea, but on fluoroscopic examination a dilated, tortuous, ascending, transverse, and descending aorta. Electrocardiogram shows a marked left preponderance, bigeminus, T-waves inverted except in the third lead (Fig. 221). Diagnosis: Generalized atherosclerosis, atherosclerotic kidney with arterial hypertension, atherosclerotic aortitis with anginoid pains, auricular extrasystoles with bigeminus.

CASES ASSOCIATED WITH HEART MUSCLE FAILURE

A certain number of these cases of coronary disease present themselves with the outstanding feature of heart muscle failure, namely, dyspnea or orthopnea, edema of the extremities, cyanosis, enlarged tender liver, ascites, etc. Quite frequently these cases occur in women, often with severe arterial hypertension, in whom some evidence of syphilis can often be demonstrated. The following 3 cases are examples of this type:

Mrs. H. E., fifty years of age, entered the hospital February 16, 1925, referred by Dr. L. E. Greensfelder. For ten months she had had severe fainting spells in which there were no premonitory signs, no convulsions, the unconsciousness lasting as long as one hour. There was often vomiting after regaining consciousness. She had three spells in ten months. She complained of insomnia and of dyspnea on exertion and orthopnea at night. She had a cholecystectomy five years before. The left radial pulse was larger than the right. The left heart border was 15.5 cm. from the midsternal line, the right 5 cm. The second aortic sound was markedly accentuated and there was a soft systolic murmur at the apex. There were occasional extrasystoles. The liver was enlarged and tender; there was moderate edema of the ankles. The blood-pressure varied from 210 to 240 systolic and 110 to 130 diastolic. The blood Wassermann was positive. The white count was 12,700. The electrocardiogram showed again a typical inversion of T in the first lead with a left preponderance (Fig. 222).

The next case of this type is Mrs. B., a widow of forty-two years. For the past few months she had noticed swelling of the ankles, and dyspnea, especially on climbing stairs. Her systolic
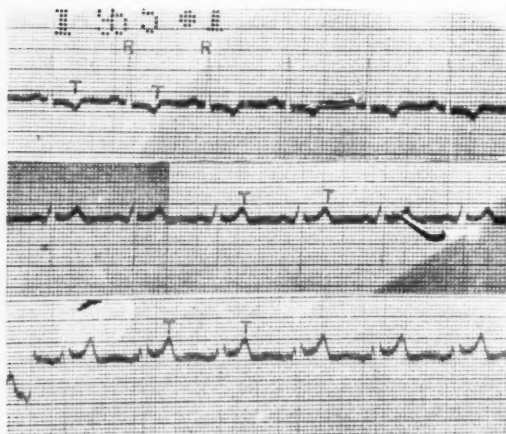
Fig. 222.—Mrs. H. E., age fifty. Recurrent "fainting spells" for ten months. Cholecystectomy five years previous. Obesity, arterial hypertension, dyspnea, edema of the ankles, positive Wassermann, inversion of T-wave in Lead I. Left ventricular preponderance.
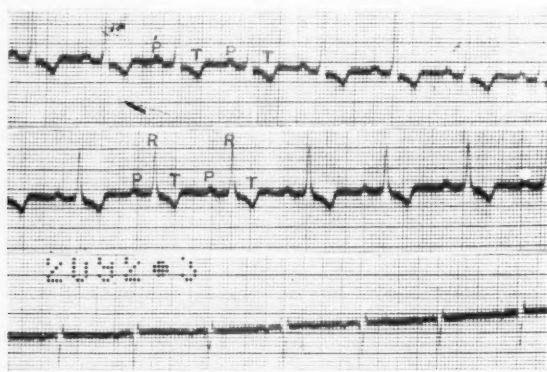
Fig. 223.—Mrs. H. B., age forty-two, widow. Dyspnea, ankle edema, arterial hypertension, syphilitic aortitis. Wassermann four plus; marked inversion of T-wave in Leads I and II. Left ventricular preponderance.

pressure was constantly over 200, diastolic over 100 mm. Hg. The liver was enlarged and slightly tender. The heart was enlarged both to the right and left, with soft systolic murmurs at

both base and apex. *x*-Ray examination revealed a dilated aorta. Wassermann was 4+. Electrocardiogram showed an inversion of T-waves in Leads I and II (**Fig. 223**).

A similar case is that of Mrs. L. S., age fifty-five years, with a blood-pressure as high as 210/100 and a left heart border of 14.5 cm. and right 3.5. In August, 1920 she had hot flashes and dizzy spells for a few weeks, with a slight swelling of the ankles and a systolic blood-pressure of 230, diastolic 110. Electrocardiogram made in November, 1920 shows again a typical in-
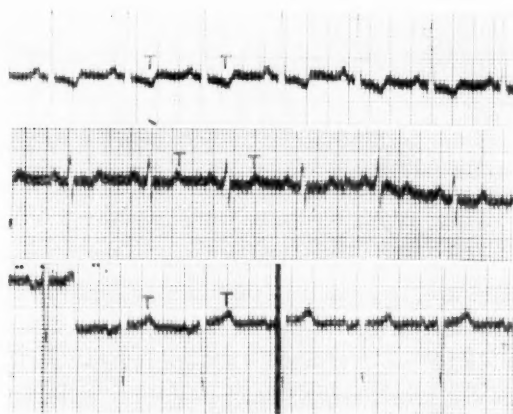
Fig. 224.—Mrs. L. S., age fifty-five. "Hot flashes and dizzy spells"; blood-pressure 230, ankle edema. Inversion of T-wave in Lead I. Left ventricular preponderance.

version of T in the first lead with a left preponderance (Fig. 224).

There is not a great deal to say about this type of case except to comment on the fact that the picture that these patients present is chiefly that of heart muscle failure with little precordial or abdominal distress. However, the fact that they showed signs of heart muscle failure and the typical inversion of the T-wave, of the electrocardiogram, justifies one in diagnosing a rather wide-spread myocardial degeneration, probably on the basis of disease of the coronary vessels.

ACUTE CORONARY OBSTRUCTION—"ACUTE INDIGESTION"

I now wish to describe an incident in these cases of coronary involvement which is so typical and so dramatic that once seen it can scarcely be forgotten, and one which it is important to appreciate and define from other somewhat similar disease syndromes. I refer to the crises or attacks of acute coronary obstruction which often occur in the course of the more chronic disease of the coronary vessels. Perhaps the most striking event in these attacks is the excruciatingly severe epigastric or precordial pain, often lasting hours and days, of greater severity and of much longer duration than the usual attack of so-called angina pectoris. This attack of pain, sometimes not unlike a protracted severe gall-stone colic and often difficult to differentiate from it, is often associated with prolonged nausea and vomiting, with collapse or complete prostration, and at times sudden death; with ashen, leaden gray countenance, with rapid, weak, thready pulse, with a sudden drop in blood-pressure even though previously high, with often later a to-and-fro friction-rub over the precordium; often with extremely enlarged and tender liver; sometimes with jaundice and usually with fever; usually with a leukocytosis within twelve to eighteen hours often as high as 15,000, 20,000, or 25,000, as first described by Libman.[8] Sometimes these patients die in the course of a few hours of the first attack, or even immediately "drop dead," apparently without pain. Some have temporary recovery for a week or two or even a number of months or years. Apparently the immediate result, whether death or temporary or permanent recovery, depends on the size and importance of the vessel obliterated, the degree and site of the heart muscle involved, and the ease and speed of the establishment of a collateral circulation. While a rapid weak pulse is the rule there may be found one of the various forms of arhythmias, paroxysmal tachycardia, auricular fibrillation, partial or complete heart-block, ventricular fibrillation, etc. The attack of coronary obstruction may be the first accident calling attention to disease of the coronary circulation or it may be the finale of a long series of premonitory symptoms. A case in point is the following:

Mr. M. B., referred by Dr. R. S. McCaughey, of Danville, Illinois, a merchant, sixty years of age, had suffered from pressure over the region of the heart and dull pain coming on after a heavy meal and when the stomach was filled with gas. It was not present with light food or liquids. It was relieved by belching. While being observed in the hospital for a suspected abdominal lesion, and while undergoing x-ray and various laboratory examinations, he was suddenly seized with an exacerbation of the dull pain in his chest three days after admission to the hos-
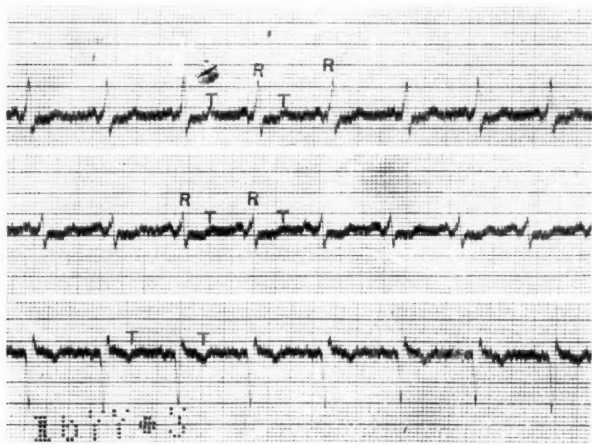
Fig. 225.—Mr. M. B., merchant, age sixty. "Dull pain in the stomach following heavy meals, relieved by belching." Sudden death from coronary obstruction during gastro-intestinal examination. Left preponderance. Somatic tremors. Typical T-wave inversion absent.

pital. The heart tones were faint and rapid, the radial pulses were unequal, he was orthopneic, pale, and slightly cyanotic; the heart tones were almost imperceptible, the rate 120; systolic blood-pressure 98, diastolic 62; white count 16,000. He died within three hours of the onset of the severe chest pain. Autopsy was not permitted. An electrocardiogram taken a few days before death shows a left preponderance, some slight inversion of the T-wave in the second and third leads, but no striking typical inversion in the first lead (Fig. 225).

Dr. H. R., age forty-four years, for seven weeks complained of pain in the left chest, radiating down the left arm. He was a practising physician and was apparently in perfect health until several weeks before. On April 2, 1925, while driving his car to the theater, he was suddenly seized with excruciatingly severe pain over the left chest which caused him to stop immediately and sit still. The pain was described as a "cramp of the heart," as if the heart were being squeezed in a vice, radiating down the left arm. The attack lasted twenty minutes and was accompanied by considerable prostration. After the attack he resumed driving, only to suffer a similar attack at the theater, which lasted one hour. Thereafter he got transient recurrent attacks of a similar nature once or twice a day, often preceded by slight exertion, such as eating, driving, walking, etc. On April 5th, while evacuating his bowels, he was taken with a terrific attack of severe pain over the heart, radiating down the left arm, which lasted four hours. He did not remain quiet during this attack, but tossed about constantly. The two physicians who were called thought he could not survive. The physical findings in this patient were strikingly similar to those already described. On May 19th, following a prolongation of one of his attacks of pain, he died. Autopsy was refused. No electrocardiograms were taken.

Another patient of this type is Mr. H. McB, seventy years of age, seen in consultation with Dr. H. Schiller. Twelve days before, while walking, he was seized with severe pain in the chest, radiating to the shoulder and neck, which lasted three or four days. His blood-pressure, which had been previously high, dropped to a systolic pressure of 92 and diastolic of 76. His pulse was weak and rapid, occasionally irregular, with a rate of 110. Under moderate amounts of strophanthis and digitalis the radial pulse suddenly dropped to 42 and 22. Ten grains of diuretin four times a day were without effect. His fever mounted to 100.6 and 100.8, leukocyte count was 22,000. An interesting feature in this patient was the fact that although he was practically in constant pain, unrelieved by morphin and other opiates, he refused to lie in any position except flat on his ab-

domen with his face buried in the pillow, stating that any other position was quite unendurable. Also instead of the usual ashen, pale, leaden-gray countenance, with mositure and cold clammy perspiration, this patient's face constantly was red, injected, and florid. Death occurred ten days after the onset.

PATHOLOGY

The hearts of patients dying from coronary disease are usually enlarged more to the left than the right; the myocardium is often markedly thickened, with particularly increased weight of the left ventricle, often with grayish-white areas of myocardial degeneration and fibrosis. The various branches of the coronary vessels are sclerotic, thickened, and tortuous. The mouths of the coronary arteries may be partially occluded, particularly in cases of syphilitic aortitis. Section of the coronary vessels often reveals the site of thrombosis with the thrombosed area partially or completely obstructing the lumen of the free or partially sclerosed vessel. Large or small areas of infarction of the left ventricle in the area supplied by the thrombosed vessel may often be seen, the infarcted portion varying from a myomalaceous area, with hemorrhages, to a small dilating aneurysm or finally a fibrosed scar.

DIAGNOSIS

The clinical diagnosis of these patients may usually be reached from the findings I have described, namely, the history, physical findings, negative x-ray findings in suspected abdominal disease, and, last, the electrocardiogram. The most striking feature of the latter, as I have mentioned a number of times, is the negativity of the T-wave in all leads or in a single or several leads. Of course, this negativity of the T-wave must be found unassociated with digitalis administration, for, as is generally known, digitalis may cause a quite similar inversion. We* have recently been interested in attempting to reproduce inversion of the T-wave experimentally in animals, and for this

* Experimental Coronary Embolism: Hamburger, Priest, Bettman, and Howard, American Journal Medical Science. (In press.)

purpose injected the anterior descending branch of the left coronary artery of dogs with a suspension of lycopodium spores. In Fig. 226 may be seen the normal electrocardiogram of the dog as a control, showing upright T-waves in all leads, and the electrocardiogram of the same animal a month later, following the
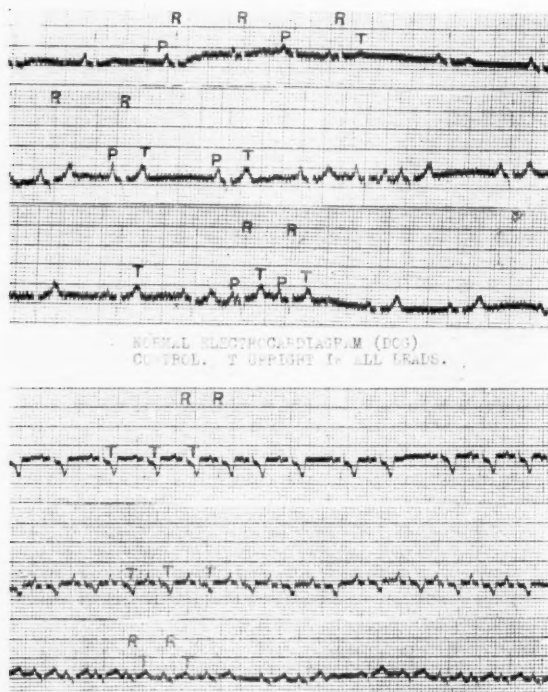
Fig. 226.—Experimental coronary embolism. Typical inversion of coronary T-wave in Leads I and II following injection of suspension of lycopodium spores in coronary circulation (dog).

injection of a suspension of lycopodium spores, showing a typical inversion of the T-wave in the first and second leads (Fig. 226). Comparing these changes with those of the clinical cases, one is struck by their similarity. Autopsy six weeks later revealed fibrous scars in the region of the left ventricle, in the area sup-

Fig. 227.—Experimental coronary embolism. Region of apex of left ventricle showing replacement of heart muscle-fibers by fibrous tissue and lycopodium spores (right); capillary overfilled with spores (left).

plied by the vessel injected, and microscopic examination showed an area composed of a mixture of fibrous scar, heart muscle, and lycopodium (Fig. 227). Also in Fig. 227 can be seen one of the small vessels overfilled with spores.

TREATMENT

The treatment may be divided into treatment of the symptoms due to chronic coronary vessel disease (sclerosis) and the treatment of the acute attack. The treatment of the former consists of general instructions as to diet, exercise, and the administration of small amounts of iodids over a long period, the limitation of tobacco, coffee, etc., moderation in diet, high seasoning, stimulants, etc., the occasional administration of digitalis in those cases associated with heart muscle failure, the administration of diuretin, nitrites, euphyllin, etc.* The treatment of the acute attack resolves itself largely into the administration of morphin for the relief of pain, the administration of caffein in fairly large amounts intravenously at times, or of other purin derivatives, diuretin, euphyllin, theobromin, etc.

PROGNOSIS

A great deal of interest centers around the question of the prognosis in these conditions, particularly the prognostic significance of the inverted T-waves. While one hesitates to be dogmatic in clinical medicine, there is little question that the outlook in many of these cases is not good, particularly when inversion of the T-wave remains permanent, and, further, when the inversion of the T-wave is associated with other electrocardiographic evidence of extensive myocardial damage, notably the finding of arborization or bundle branch block. However, to put a definite time limit of eight, ten, twelve or more months, just because of these electrocardiographic findings, I feel is not

* Treatment of the abdominal symptoms, which are the major complaints of these patients, is best carried out with careful attention to size and frequency of meals, regulation of bowel function and the administration of such simple remedies as baking soda, milk of magnesia, aromatic spirits of ammonia, peppermint, charcoal, etc.

possible. In the cases presented there are several who show the presence of T-wave inversion who have been engaged in fairly active lives for a period of five years and longer.

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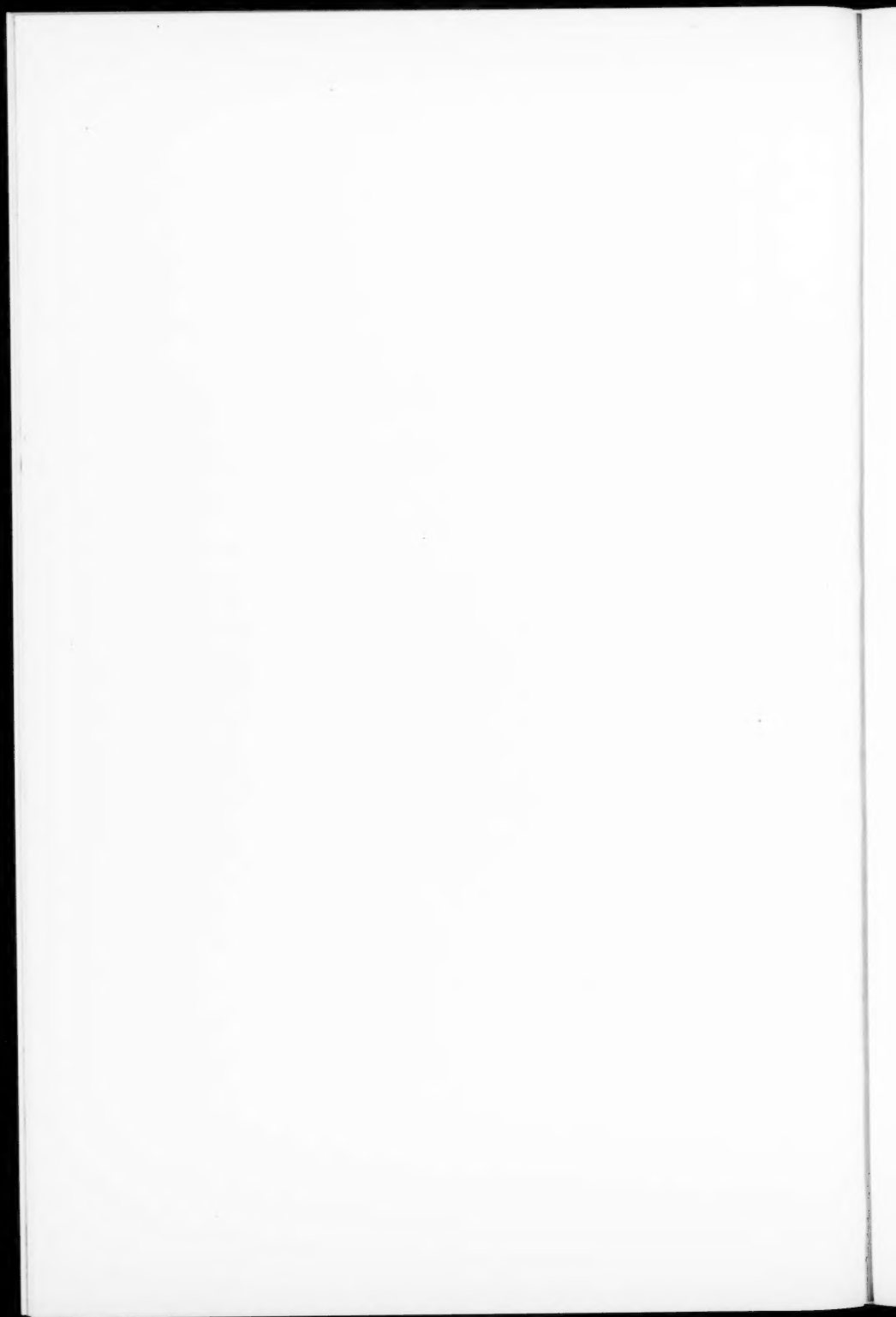
MIDDLE-EAR AND MASTOID INFECTIONS

THE subject of our talk today is middle-ear and mastoid infections. Primary middle-ear or primary mastoid infection is, as we know, rarely encountered. Practically all of this class of
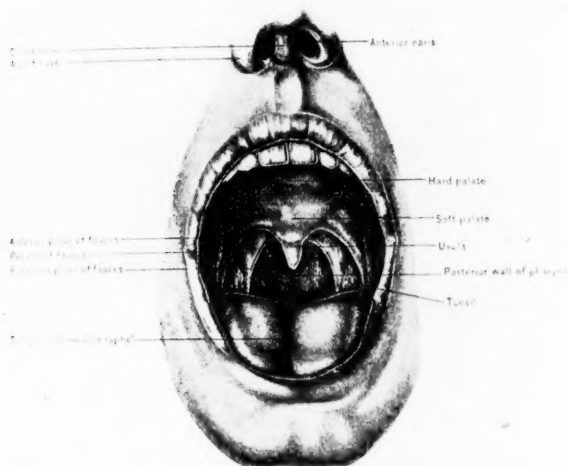
Fig. 228.—Structures of the throat.

infections are secondary ones, the primary focus originating in the throat, nose, nasal accessory sinuses, nasopharynx, or the eustachian tube. I have found it of advantage in a clinical and pathologic study to regard these several anatomic units—the throat, nose, nasal accessory sinuses, nasopharynx, eustachian

tube, middle ear, and the mastoid—as a *composite structure.* The seven units of the group function in a reciprocal way and are intimately related physiologically and pathologically. It will be of value, I am sure, to briefly review, with the aid of a few stereoptican slides, some of the outstanding anatomic and physiologic features of these units before entering upon the subject of my talk.
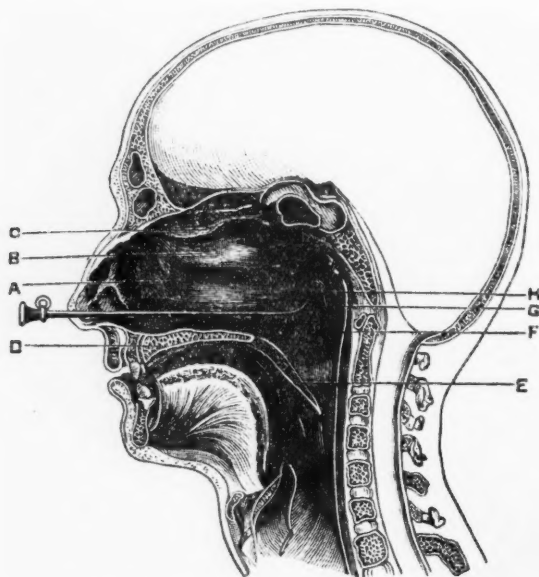
Fig. 229.—Vertical section of the nasopharynx with the catheter introduced into the eustachian tube: *A*, Inferior turbinated bone. *B*, Middle turbinated bone. *C*, Superior turbinated bone. *D*, Hard palate. *E*, Velum palati. *F*, Posterior pharyngeal wall. *G*, Rosenmüller's fossa. *H*, Posterior lip of the orifice of the eustachian tube. (Politzer.)

The first slide (Fig. 228) I show you is that of the throat. There are three structures in the throat which concern us *apropos* of our subject of middle-ear and mastoid infections, namely, the tonsils, the pharyngeal opening of the eustachian tube, and the lymphoid tissue of the nasopharynx, the so-called adenoids.

The tonsils are located on the lateral wall of the oropharynx, in the sinus tonsillaris, and are protected in front and behind by a mucomuscular septum, the anterior and posterior pillars formed by the palatoglossus and palatopharyngeus muscles.

The second slide (Figs. 229, 231) presents the lateral wall of the throat; a eustachian catheter has been passed through the inferior meatus of the nose into the pharyngeal opening of the
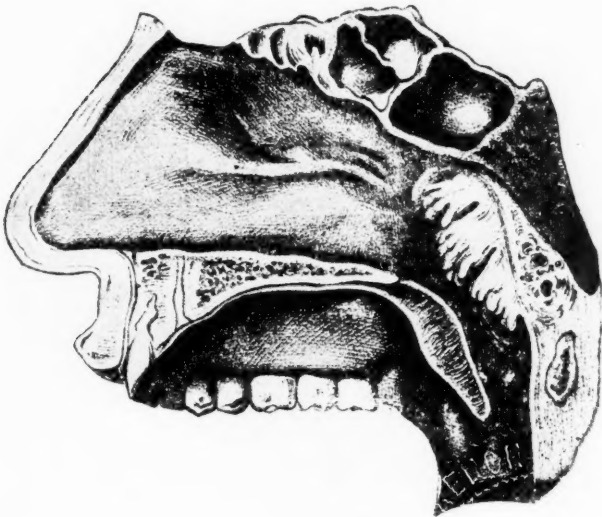
Fig. 230.—Anteroposterior section of the head of an adult, showing the situation and gross structure of hypertrophy of the lymphoid tissue of the nasopharynx (Zuckerkanal).

eustachian tube. The third slide (Fig. 230) exhibits adenoid tissue in the nasopharynx.

The particular structure of the throat which is most vulnerable to ear integrity is the pharyngeal opening of the eustachian tube. One is impressed in viewing these slides by the intimate relation of the pharyngeal tubal opening to the tonsil and to the nasopharyngeal lymphoid tissue. The tube is the connecting link between the throat, nasopharynx, and nose below and the

middle ear and mastoid above. It is about $1\frac{1}{4}$ to $1\frac{1}{2}$ inches long,
partly osseous and partly cartilaginous, lined with mucous mem-
brane, paved with ciliated epithelium, the ciliary motion being
directed toward the throat; in the infant it is wider, straighter,
and more horizontal than in the adult; its function is to venti-
late and drain the middle ear and mastoid. Ordinarily its mem-
branous walls are assumed to be contracted; during deglutition,
however, the tensor and levator palati, assisted by the palato-
pharyngeous, open its pharyngeal orifice, permitting air to enter
the tube and supply ventilation to the middle ear. In order that
the tube performs its functions properly its patency must be
maintained and its cilia remain undamaged. As a result of tubal
blockade two things happen: first, the blood in the middle-ear
tissues absorbs the oxygen content in the middle-ear cavity,
creating a vacuum or negative pressure, which produces a local
tissue hyperemia; second, the retained secretions undergo de-
composition, tissue resistance is lowered, and a favorable soil
is thus created for the deposition of pathogenic micro-organisms.
If the difficulty, for example, be a tonsillitis, all the contiguous
structures, in a general way, share in the inflammatory invasion;
in particular, the assistance, contributed by the palatopharyn-
geus in opening the tubal pharyngeal orifice, becomes impaired,
and as a result of all these factors a tubal blockade is produced.
In children adenoids are a frequent cause of tubal blockade,
the blockade resulting more from the actual obstruction they
produce than from an inflammatory process.

The anatomic features which I have enumerated as well as
our clinical experiences clearly suggest that a sore throat or
adenoids should not be regarded as a trivial thing. Patients,
particularly children, who have adenoids and who are, as the
saying goes, "subject to sore throat," demand attention. Each
sore throat exposes such patients to the danger of middle-ear
infection, and each middle-ear infection to the danger of a mas-
toid infection.

The next slide (Fig. 231) presents a cross-section of the nose.
We note the nasal septum, the turbinates, their corresponding
meatuses, the frontal sinuses, ethmoids, antra, orbits, and their

relations.   The functions of the nose are four—respiratory, ol-
factory, phonatory, and gustatory.

Clinical experience has demonstrated that nose and sinus in-
fections are exceedingly common.   My own experience is that,
in adults, I encounter more cases of nasal and sinus difficulty than
I do those of the throat.   Our anatomic studies of the nasal ac-
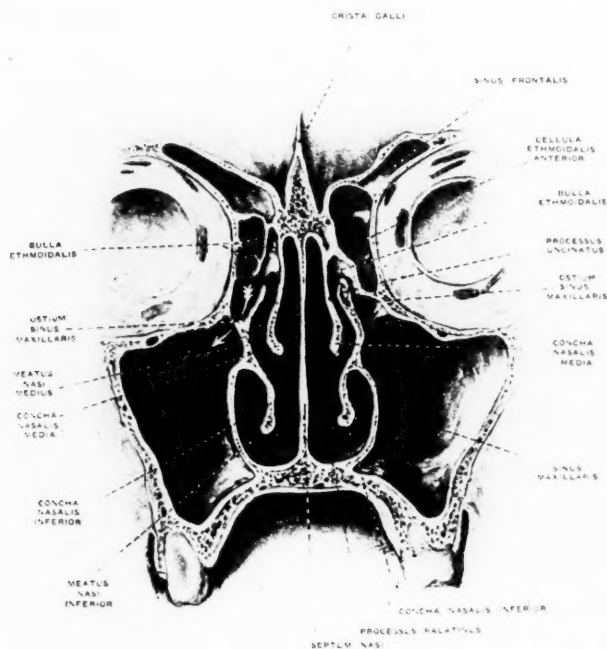
Fig. 231.—Coronal section through the nose and orbit.

cessory sinuses show us their exceedingly intimate relations with
each other, with the nose, the nasopharynx, the throat, and,
through the eustachian tube, with the middle ear and mastoid.
Their exact function is not well established.   Clinical experience
teaches, however, that their drainage outlets must not be dis-
turbed.

The most vulnerable region of the nose is the middle and superior meatus, which I am fond of referring to as the "nasal danger zone." All the sinuses have their ostia or drainage outlet in this region. It is probable that the majority of sinus infections are primarily induced by obstruction of their normal drainage outlets, usually in the form of septal deflections, hypertrophy of the turbinates, septal spurs, etc. The blockade thus produced results in retention of sinus secretion, destruction
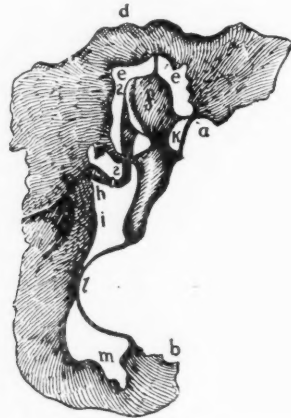
Fig. 232.—Coronal section through the tympanum: *a*, Extremity of the upper, *b*, extremity of the lower, bony wall of the meatus; *d*, tegmen tympani; *e, e*, attic, external portion, internal portion; *f*, malleus and superior ligamentum mallei; 2, incus; *h*, stapes within the fenestra vestibuli; *i*, promontory; *k*, Prussak's space; *m*, hypotympanic recess (cellar); 1, scar in the lower half of the drumhead in apposition with the promontory; 2, incudostapedial junction. (After Brühl-Politzer.)

of their cilia, and a lowered tissue resistance, which eventuates in the deposition on the favorable soil of pathogenic bacteria with the ultimate production of a purulent sinusitis.

The next series of slides present the ear and mastoid.

The Middle Ear (Tympanic Cavity; Tympanum).—The tympanic cavity is the air space between the tympanic orifice of the eustachian tube and the mastoid antrum. Its lining membrane, mucous membrane, is continuous with that of the eu-

stachian tube, extends to the antrum and mastoid cells, and is
covered with ciliated epithelium, whose wave-like motion carries
the secretion to the eustachian tube.   Its external wall is the
drum, with bony extensions above and below; internally is the
labyrinth; anteriorly, the eustachian tube opening; posteriorly,
the aditus ad antrum, its upper wall (tegmen tympani) forms
part of the middle cranial fossa, its lower wall is in close relation
to the jugular bulb.
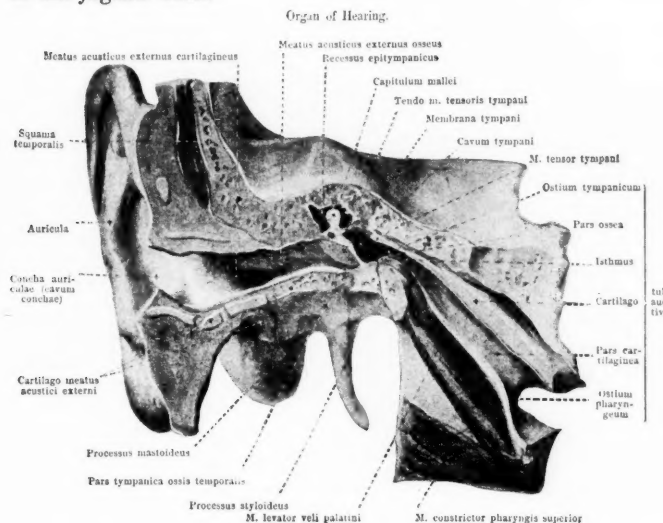
Fig. 233.—General view of the right external ear and middle ear, looked
at from in front and externally.   The external ear has been opened by a
frontal section; the tympanic cavity and eustachian tube have been opened
by a vertical section carried obliquely, lateralward, and dorsalward (Spalte-
holz).

The *tympanic space* (Figs. 232, 233) itself is an irregularly
wedge-shaped cavity.   Its roof corresponds in position with the
floor of the middle cranial fossæ.   It is normally divided into
three parts:

The *vault* (attic, the epitympanic space), which lies above
the level of the short process of the malleus, the *atrium* is the
area between the upper and lower margins of the membrana

tensa, the *hypotympanic space* (cellar) lies below the level of the floor of the bony canal. In order properly to appreciate the clinical evolution of the different types of middle-ear infections it is important to stress the anatomic peculiarities of the atrium and the attic. These may be summarized as follows: The *atrium* is lined by a thin, firmly adherent membrane, it is closed externally by the relatively unyielding drum; it is practically walled off from the attic by the neck and short process of the
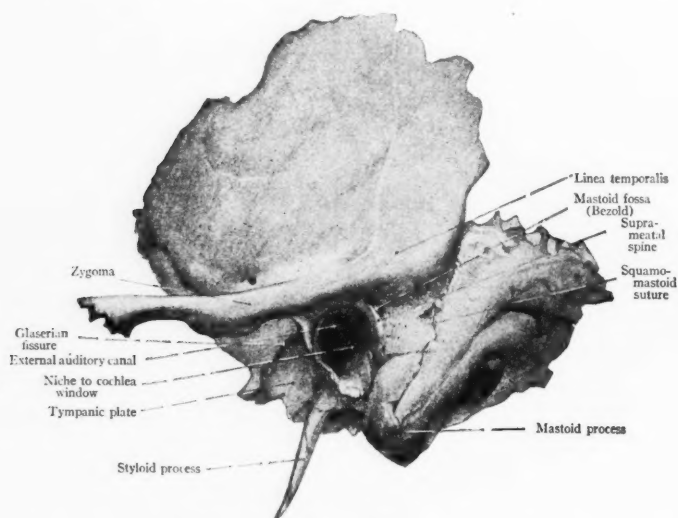
Fig. 234.—Left adult temporal bone, showing landmarks (Barnhill and Wales).

malleus, the lower anterior extension of the body of the incus, the anterior, external, and posterior ligaments of the malleus and the mucous membrane which surrounds them; on its anterior wall the eustachian tube leads to the nasopharynx. The *attic* is continuous directly with the mastoid antrum, its lining membrane is not closely adherent, but hangs in folds from the tympanic roof, and these folds divide its space into compartments.

The drum membrane, as a whole, is divisible into two parts: the *membrana propria* (or tense membrane) and *Shrapnell's membrane* (or the membrana flaccida).

The *ossicles* are a system of bones, three in number, the malleus, incus and stapes, which are housed in the middle ear, articulate, and connect the drum with the labyrinth.

The *mastoid process* (Figs. 234–236) in the adult is a conical bony projection, pointing downward, and located behind the external auditory meatus. The external surface is known as
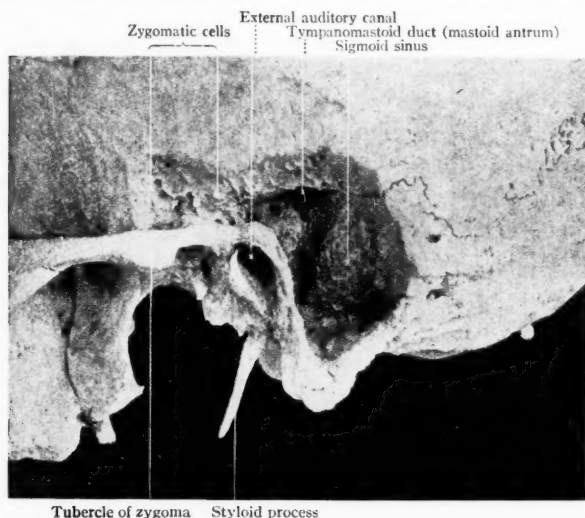
Fig. 235.—Skull showing great development of zygomatic cells (Barnhill and Wales).

the cortex; the internal surface, as the inner table; between the two surfaces are the mastoid cells. On the inner side of the process is the digastric fossa which gives attachment to the posterior belly of the digastric muscle. The osseous wall at this point is frequently as thin as paper, and it is an exceedingly vulnerable point for pus to break through and burrow into the muscles and deep fascia of the neck. The inner table presents a deep groove, the fossa sigmoidea, which lodges a portion of the lateral sinus.

Three types of mastoid cells are recognized; the *pneumatic*, containing numerous cells; the *diploic*, containing only the antrum, diploic tissue making up the rest of the structure; the *sclerotic*, containing only the antrum, a dense eburnated mass of osseous tissue filling the process. One cell is invariably present in every process, namely, the antrum. In the adult the antrum is somewhat kidney shaped, is located behind the tympanus, and communicates by the aditus ad antrum with the tympanic vault. The depth of the antrum in the adult varies greatly; rarely is it less than $\frac{1}{2}$ inch below the surface. The
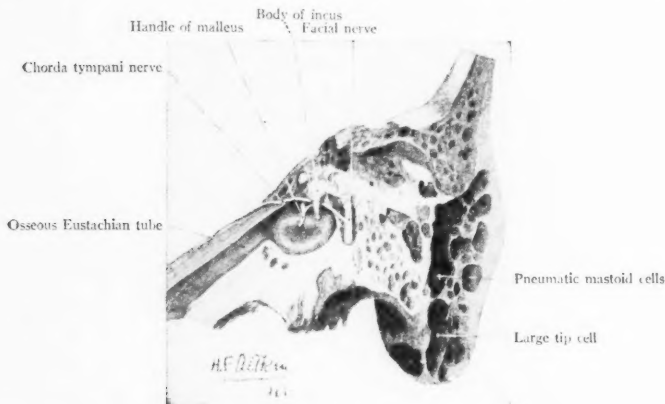
Fig. 236.—Right temporal bone. Vertical section through middle ear, viewed from within (Barnhill and Wales).

roof of the antrum is a thin plate of bone continuous with the roof of the attic, the tegmen tympani, and forming part of the floor of the middle cranial fossa. The mastoid cells connect with one another, with the antrum and with the middle ear through the aditus, and are lined with a continuation of the mucous lining of the middle ear. This lining constitutes the periosteal as well as the mucous covering, is highly vascular, and provides nourishment for the osseous structures it covers.

At birth the infant presents no distinct mastoid process, only a small, undeveloped tuberosity. This contains a single cell,

the *antrum,* which is usually superficially placed, directly above and a little behind the external auditory meatus. The intimate and relatively large connection of the mastoid antrum to the tympanic vault, as well as the superficial location of the antrum and softness of mastoid structure in the young, especially conduces to extension of infections from the middle ear and perforation of mastoid cortex with consequent production of subperiosteal abscess behind the auricle. Thinness of the mastoid wall in the region of the digastric fossa invites perforation with the development of abscess in the soft tissues of the neck. The walls of the facial canal in infants are not only very thin, but not uncommonly are incomplete and expose the nerve to inflammatory exudations, which explains the frequency of facial paralysis in children. The presence of mastoid cells on the posterior wall of the external canal explains the mastoid symptoms which are the almost constant accompaniment of an acute otitis media. In mastoids of the pneumatic type a purulent process is likely to progress more rapidly than in those of the sclerotic type.

The series of slides which I have just shown you emphasizes the intimate anatomic relation existing between the throat, nose, sinuses, eustachian tube, middle ear, and mastoid. This association is so intimate that a disease in one of these structures is exceedingly likely to affect any one or all of the other structures comprising the group.

Having presented this side of our subject as a preliminary or prologue, so to speak, we will now speak of middle-ear and mastoid infections. It is possible in our brief time limit to discuss only acute otitis media and acute mastoiditis. Time also will not permit a consideration of the surgery of the mastoid.

ACUTE OTITIS MEDIA

First let us discuss *acute otitis media.* Some difference of opinion exists among otologists regarding the classification, the differentiation, etc., of tubotympanic congestion, acute catarrhal otitis media, and acute purulent otitis media, but such divergence of opinion does not apply to their etiology, since the etiology of

all of these difficulties is practically identical. Viewed in a practical sense, therefore, it is permissible to discuss this group under the one title, viz., acute otitis media, pointing out, as the occasion demands, the essential differences which characterize them clinically.

It may be definitely stated as a principle that all patients who suffer from embarrassed nasal respiration due to nasal obstruction, such as septal deviation, nasal spurs, polyps, etc., or hyperplastic, hypertrophic inflammatory types, causing enlargement of the turbinates, or those suffering from atrophic nasal disturbances causing abnormal enlargement of the nasal spaces, are candidates for acute middle-ear infections. Disease of the nasal sinuses likewise may affect the nasal spaces, their contiguous structures, and are common causative factors in the production of middle-ear infection. In children "adenoids" are a very common cause.

The most frequent exciting cause is the common "cold in the head." Swimming, blowing the nose violently, especially when a patient has a sore throat, and improper use of nasal douche are very frequent causes.

Middle-ear infection is exceedingly common as a complication of the exanthemata, particularly scarlatina, measles, diphtheria, the flu, pneumonia, etc. It should be emphasized that ear lesions complicating scarlatina are prone to produce destruction of ear structures causing serious impairment of hearing, etc.

Symptoms.—It is important to distinguish between infection of vault and infection of the atrium (Figs. 232, 233). We recall from our anatomic studies that there is a considerable difference in the anatomy of these two spaces which influence the evolution of a middle-ear infection in a very important way. To summarize, these anatomic differences are as follows: The *atrium* contains little connective tissue, its lining membrane is thin and closely adherent; its mechanical separation from the vault conduces to limitation of the infection and early rupture of the drum, with drainage of the infective material. The *vault* is directly connected with the antrum; its lining membrane hangs in folds from its roof, and these, with the various ligaments and portions

of the ossicles it contains, serve to divide its space into compartments, and also separates it from the atrium. Hence, in infections of the vault, the purulent material finds an easier route of exit through the antrum than the relatively strong drum membrane provides and, in consequence, earache does not come as suddenly, is not so severe at the outset, drum perforations are more delayed, the antrum is early infected, and mastoid involvement is more likely than in infections confined to the atrium.

The following are the principal symptoms and signs: stuffiness and fulness in the ears, earache, temperature, ear discharge, tinnitus, impairment of hearing, changes in the drum varying from slight localized redness to intense generalized injection, bulging and perforation, and antrum tenderness.

Of the specific symptoms the outstanding one is *earache.* The earache is a *pressure* or *tension* symptom, and is due to the engorgement of the tympanic vessels and pressure by the retained fluid in the small tympanic space.

Naturally its severity is influenced by the temperament of the patient, but there is perhaps no pain of any disease more exhausting or distressing. Hearing at the onset is more or less disturbed. An early symptom is stuffiness of the ear.

The drum changes depend upon the stage of the disease and the site of infection—atrium or vault. It may be only slightly reddened at the outset; later very much reddened and bulging. Practically all cases suffer at some stage of the disease from stuffiness of the ear, tinnitus, and impaired hearing in varying degree. Temperature elevations of moderate degree is usual, but it is not uncommon for it to remain normal or only slightly elevated. As might be expected from its intimate relation to the middle ear, tenderness over the antrum is practically always present, particularly if the vault is the seat of the infection. Ear discharge follows as a result of perforation of the drum or after it has been incised. It may be serous or purulent in character. Frequently after a drum incision little discharge appears before a day is past. The most common site of the perforation is the posterior superior drum quadrant.

Infants and young children exhibit very often considerable elevations of temperature. Since no subjective data in the young is obtainable, such suggestive data as restlessness, irritability, manipulation of the ear, sleeplessness, etc., is significant of middle-ear infection. An ear examination of such cases is a wise measure, and I strongly advocate it as a routine procedure in all infants and young children.
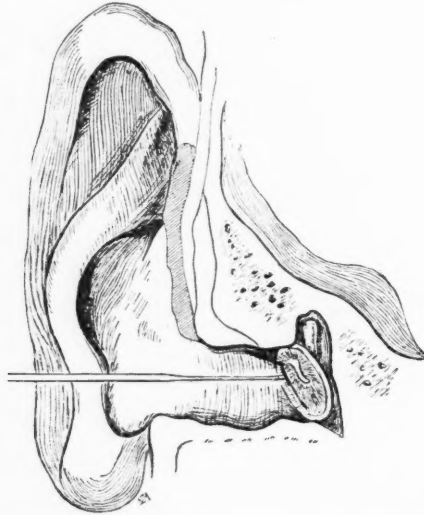
Fig. 237.—Incision of membrana tympani. The usual location and extent of incision is indicated by the vertical line along the posterior margin of the drum; to this is commonly added the incision, along the posterior wall of the meatus, indicated by the heavier horizontal line. (Modified, from Dench's Surgery of the Ear, Keen's Surgery, vol. iv.)

Treatment.—There is a so-called abortive treatment, but in the average case the physician is not usually called until the process is well advanced and the time for abortive measures has passed. The abortive measures comprise local *heat* (hot-water bag or electric pad), irrigation with warm sterile boric solution, and instillation of 10 per cent. phenolglycerin. Under no circumstances should narcotic drops be instilled in the ear.

They mask the symptoms, and if a perforation exists expose the patient to toxic influences. The abortive general measures are a laxative, light diet, and such sedatives as codein, phenacetin, aspirin, etc.

The proper treatment in the vast majority of cases is free incision of the drum which has as its object the providing of free drainage followed by efforts such as the insertion of a gauze wick in the canal or irrigation, to maintain the drainage and prevent the stagnation and accumulation of the purulent material. I am a strong advocate of early incision of the drum, and I think if it is carried out early and efficiently, especially in children, the suffering is promptly ended, hearing is conserved, a chronic process is averted, and the great danger of mastoid and intracranial infection is avoided. A local or general anesthetic may be used and the incision is not merely a puncture, but a free opening (Fig. 237). It is far better to make an early free opening than to wait for perforation. The former heals quickly and no damage to hearing results; the latter heals slowly and may cause adhesions which result in impaired hearing. The subsequent care may require special measures, such as inflation, local treatment, etc., to prevent adhesions, heal perforations, and preserve hearing.

ACUTE MASTOID INFECTION

Acute mastoiditis is practically always the result of an otitis media. All the etiologic factors previously described which play a part in the infections of the middle ear may, therefore, be included as possible causative factors in the production of a mastoid infection. Delayed or inefficient drainage of a suppurative otitis media is perhaps the outstanding cause in the majority of cases. Other factors, such as lowered vitality, consequent upon a long or particularly severe illness, etc., are undoubtedly predisposing. The diagnosis is made from a study of the history, the subjective symptoms, data obtained from examination of the middle ear and mastoid, examination of the blood, smears and culture of the ear discharge, and single or serial radiograms of the mastoid.

The symptoms and signs of mastoid infection are subject to considerable variation, and this fact must be kept strongly in mind in interpreting or anticipating the clinical progress of an individual case. Pain or discomfort about the ear and mastoid is usually an early symptom. Restlessness and insomnia, as a rule, accompany this symptom. Increase of temperature is a

Fig. 238.—Points of mastoid tenderness in acute mastoiditis. The uppermost + is over the site of the mastoid antrum, the lower + over the mastoid tip, and the posterior one is over the point of exit of the mastoid vein. The mastoid region has been shaved preparatory to operation (Barnhill and Wales).

fairly constant symptom, though cases are not rare exhibiting normal or only slight elevations. Careful analysis of the behavior of the ear discharge is of value. A profuse ear discharge unyielding to treatment, practically continuous for over a period of two weeks, is strongly suspicious of extension of the infection to the mastoid; a progressive increase of the discharge over a similar period resisting efficient treatment likewise points to

mastoid involvement; a more or less sudden cessation of the discharge, associated with ear and mastoid discomfort, is suggestive of a beginning mastoid invasion. Tenderness of the mastoid is undoubtedly the most reliable and constant symptom. Four "points of tenderness" are encountered, viz., antrum, tip, emergence of the emissary vein, the premastoid lamina (Fig. 238). The technic of eliciting these "tender points" is important. The thumb is applied with even pressure directly to the interrogated point and no "rocking" or "sliding" of the tissue is per-
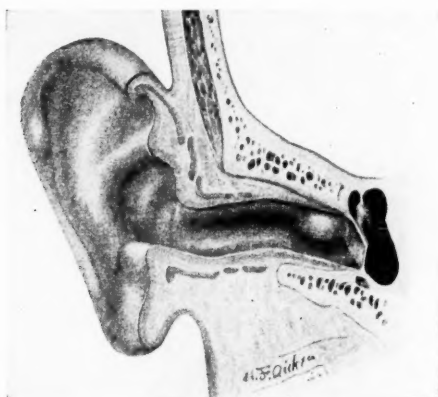
Fig. 239.—Sagging of the posterosuperior wall at the inner end of the external auditory meatus, the result of mastoid suppuration in the adjacent pneumatic cells of the mastoid. Compare location of the sagging with tumefaction of canal due to furuncle, Fig. 242 (Barnhill and Wales).

mitted. In estimating "antrum tenderness" especially the auricle must not be moved; should a furuncle of the external canal be present, the slightest movement of the auricle would elicit pain, and a wrong interpretation of the pressure test would result. The healthy mastoid serves as a basis for comparison. Of the "four tender points," the antrum tenderness is the most dependable.

The period of onset of mastoid tenderness is suggestive. With the subsidence of pain and discharge it should grow less; should it increase, it indicates an advancing mastoid infection;

if absent at the beginning of a middle-ear suppuration and it appears later, extension to the mastoid is probable. In estimating mastoid tenderness it is important to emphasize that in practically all cases of acute middle-ear infections involving the vault a certain degree of antrum infection and tenderness is present, due to the intimate anatomic relation of the antrum to the vault. This so-called "middle-ear antrum tenderness" may for a limited period, in the absence of other confirmatory mastoid symptoms,

Fig. 240.—Projection of the auricle in mastoiditis with postaural abscess. The collection of pus is above and behind the auricular attachment. Compare the condition with that produced by a rupture of pus into the digastric fossa, Fig. 241. Note also "adenoid expression" (Barnhill and Wales).

be excluded as an evidence of mastoid disease. Sagging or bulging of the posterosuperior meatal wall is regarded as an exceedingly valuable symptom, many considering it a pathognomonic symptom, demanding immediate mastoid operation (Fig. 239). Associated with this symptom is bulging of the drum and narrowing of the external canal lumen at the fundus.

Edema of the mastoid may occur as a result of extension of the inflammatory process to the soft tissues, without perfora-

tion of the cortex. A subperiosteal abscess may result from a perforation of the cortex, the inflammatory material elevating the mastoid periosteum and displacing the auricle (Figs. 240, 241). Similarly, a perforation may occur in the digastric fossa, causing a swelling below the mastoid, in the neck. This is known as Bezold's abscess. The value of a differential blood-count is not well established. It is probable that a marked leukocytosis and high polynuclear percentage is of value in connection with other confirmatory signs of mastoid involvement. The evidence
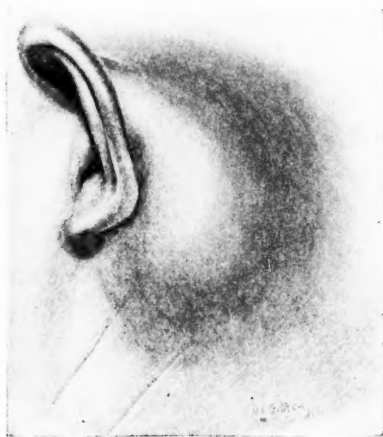
Fig. 241.—Bezold's abscess. Compare the position of the auricle and of the tumefaction with that produced by rupture through the mastoid cortex over the site of the antrum, as shown in Fig. 240 (Barnhill and Wales).

obtained from a bacteriologic examination of the ear discharge may prove of value; when Friedlander's bacilli, streptococci, Klebs-Löffler, pneumococci and tubercle bacilli are present, earlier operation is indicated than if the staphylococci or mixed infection is present.

Radiograms singly or in series I have found to be of value in doubtful cases. Progressive invasion of the mastoid can often be observed by their use over different periods.

In infancy and early childhood the diagnosis of a mastoiditis

is frequently difficult. The majority of the usual symptoms and signs are exaggerated in intensity. Temperature elevations are usually greater than in the adult, likewise the restlessness and distress; convulsions, vomiting seizures, chills, etc., are not uncommon; subperiosteal abscesses are often met with. Furuncle of the meatus is the only condition likely to prove confusing in the differential diagnosis (Figs. 239, 242). The following differential points may be noted: Traction on the auricle is painless in mastoiditis; in furuncle, acutely painful. The swelling or
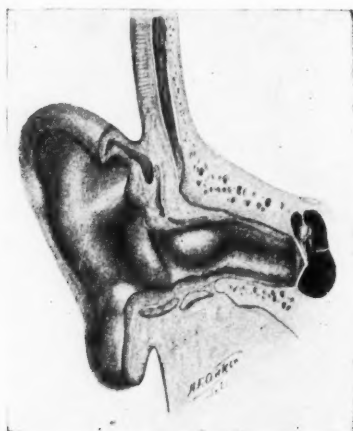
Fig. 242.—Furuncle. Note its situation on the cartilaginous meatus in the outer half of the canal. Compare this location with the sagging of the inner end of the meatus due to mastoiditis, Fig. 239 (Barnhill and Wales).

bulging of the canal wall is located near the drum on its superoposterior wall in mastoiditis; in furuncle it is located usually in its outer third. The ear discharge precedes the mastoid symptoms; in furuncle it follows the swelling, etc.

Treatment.—The treatment of an unquestioned mastoid infection is operative. Free, adequate drainage is imperative.

In selected cases, where one may hope to improve the drainage from the middle ear and, supposedly, the mastoid by a more extensive drum incision, such a procedure may be done, and in

conjunction irrigations or wick drainage, heat or cold to the mastoid carried out, and, if no contraindications arise, may be continued for twenty-four or forty-eight hours. Heat or cold applications, particularly cold, often mask the mastoid symptoms, and I feel the temporary relief they usually afford should be accepted with considerable caution. Narcotics are open to a similar objection and I employ them only in extreme cases. In my own experience, when all measures of free drainage from the middle ear have been exhausted, without definite improvement of dependable mastoid symptoms and signs, I forego further abortive measures and proceed at once to operation on the mastoid.
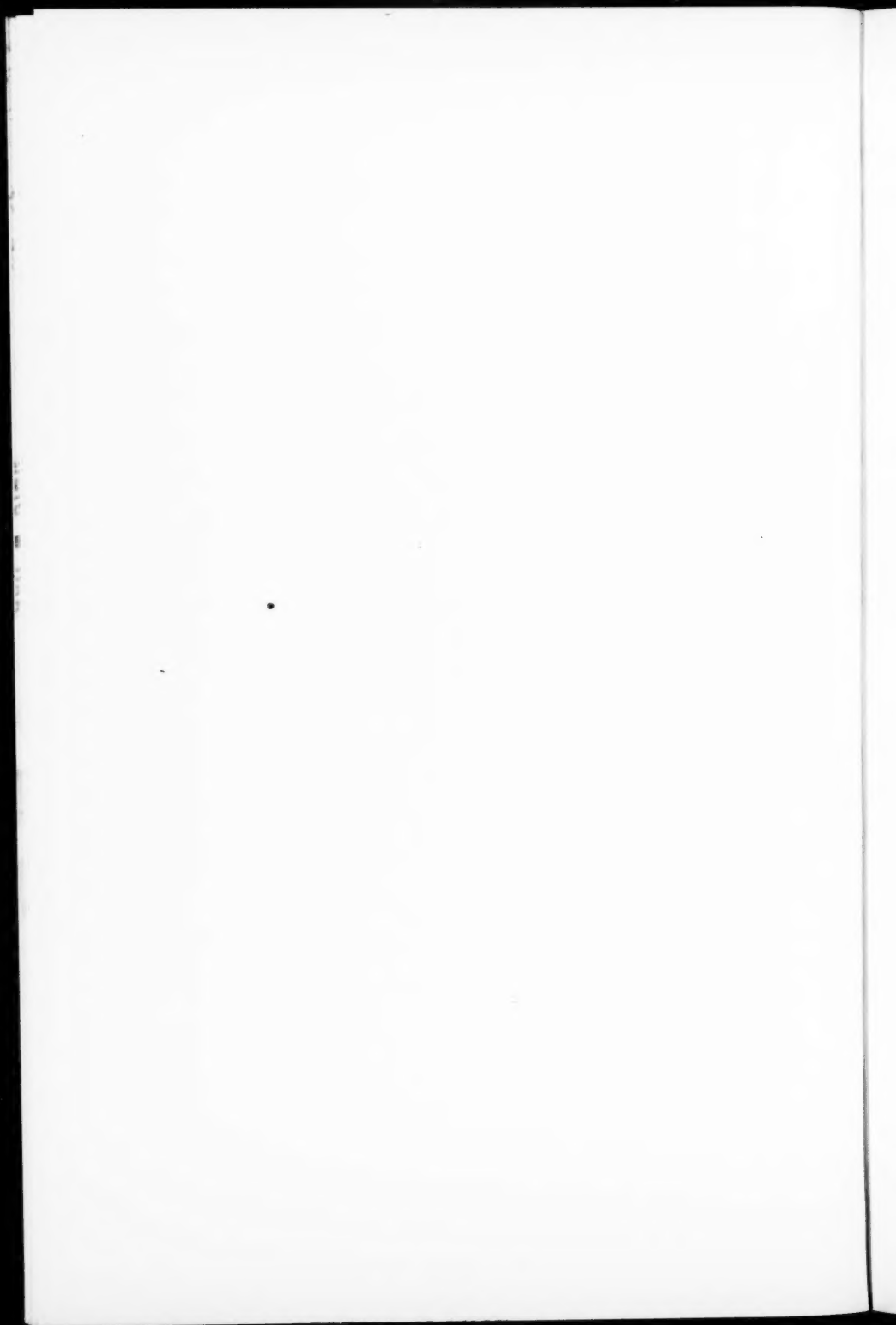
CLINIC OF DRS. ISAAC A. ABT AND ALFRED A. STRAUSS

FROM THE PEDIATRIC SERVICE OF SARAH MORRIS HOSPITAL FOR
CHILDREN AND CHICAGO LYING-IN HOSPITAL

CLINICAL STUDY OF 221 OPERATED CASES OF HYPER-TROPHIC CONGENITAL PYLORIC STENOSIS

IN previous publications we have emphasized some of the outstanding facts as to the symptoms, physical findings, and operative and postoperative results which were based upon a study of cases of pyloric stenosis. The purpose of this paper is to state our experience in diagnosis and treatment and to present some new data which we have collected from our entire series; 221 patients were treated by operation.

Age.—The greatest number of children with pyloric stenosis were between four and eight weeks old at the time they were admitted to the hospital. One infant, however, was only five days old, and another was five months old. The range in age of the cases between these two extremes is shown by the following table:

Age.	Number of infants.
Five days	1
Two weeks	4
Three weeks	34
Four weeks	52
Five weeks	27
Six weeks	24
Seven weeks	23
Eight weeks	29
Nine weeks	8
Ten weeks	13
Eleven weeks	3
Twelve weeks	4
Fourteen weeks	1
Five months	1

Sex.—Of the 221 cases studied, 161 were males and 60 were females, that is, 72.9 per cent. of the total were males and 27.1 per cent. were females.

Mortality.—In the 221 cases the operation was the usual pyloroplasty which we described in 1915. It is similar to the Rammstedt operation, modified as follows:

The mucosa is shelled out more freely from the muscularis and a plastic flap is made of the muscularis. The free edge of the attached omentum is brought over the pylorus.

Seven of the patients operated on died, giving a mortality of slightly over 3 per cent. Two of these, among the first of this series, came to the hospital moribund. Both died within twelve hours after operation. One patient died of peritonitis, the course of which was not definitely established. Two others died during the influenza epidemic in 1918 from an intercurrent influenza pneumonia three weeks after the operation. One of the fatal cases proved at autopsy to be the result of pulmonary embolus. The seventh case showed multiple atresias of the intestinal tract, one at the pylorus and another at the ileocecal junction.

As a rule patients are not operated immediately upon entrance to the hospital. We try to improve the condition of the infants before operation by giving them normal saline hypodermically and 5 per cent. glucose solution intravenously. If the condition demands it one or two blood tranfusions are given, usually before, but sometimes after, the operation.

If the infant is feeble or dehydrated, it is much better to wait one or two days (twenty-four to forty-eight hours) until the general condition is improved by the above methods.

We believe that in infants six to twelve weeks of age who present a palpable tumor our operation of modified pyloroplasty has distinct advantages over the Rammstedt. The children operated on by this method do not vomit following the procedure. They have more chance to pass successfully the critical period of the first few days than if they had a great deal of regurgitation and vomiting, as sometimes occurs after the Rammstedt.

Family History.—In practically all cases the family history was completely negative, and both parents were living and well at the time the infant entered the hospital. In all but 3 cases the health of the mother was good during pregnancy.

One mother vomited during the entire pregnancy and another vomited and had edema of the feet during the last six weeks. In 12 cases miscarriages were reported. There was one still-birth, and one infant died immediately after birth in two of the families. In the greatest number of cases the patient was either the only child or one of two children.

In one family the history stated that two older children showed an idiosyncrasy toward the mother's milk. In another family 4 older children had vomited during infancy and 2 of them died.

Maternal diseases in the 221 cases studied are summarized as follows:

Diseases.	Cases.
Tuberculosis	7
Cancer	2
Heart disease	2
Rheumatism	1
Nephritis	1
Diabetes	1

Personal History.—Birth history: In 8 cases this information was not obtained. Of the remaining 213, 210 were full term and 3 were premature; 181 were normal deliveries, 30 were instrumental, 1 was delivered by cesarean section, and in 1 case there was prolapse of the uterus. In this latter case the baby was delivered with two loops of cord around the neck. Postnatal complications occurred in 5 of the 213 cases. In 3 there was slight asphyxia. Slight bleeding of the navel for six or seven days occurred in 1, and in another the clavicle was fractured during delivery, with resultant paralysis of the right arm which persisted for ten days.

Previous Illnesses of the Infant.—One infant had convulsions and severe stomatitis at the age of nine days, but with no recurrence. Three had very severe jaundice from the second to

the seventh weeks of life. One had a pustular eruption, chiefly on the trunk, which lasted from the second to the seventh week.

Birth weight: In 92 cases the birth weight was not known. In the other cases it ranged from 6 pounds to 11 pounds, 8 ounces. Most of the children weighed between 7 and 8 pounds.

Feeding: Before admission to the hospital 119 of the infants received breast milk exclusively, 101 received breast milk and some complementary feeding, and 1 was fed artificially with no breast milk.

Onset and Course.—Vomiting was a symptom in all of the 221 cases. It appeared most frequently during the second or third week of life.

Onset of vomiting:

Age.	Cases.
At birth	10
First week	10
Second week	68
Third week	60
Fourth week	30
Fifth week	17
Sixth week	17
Seventh week	6
Eighth week	3

In 198 cases the vomiting was known to be projectile in character. In one case it was not of this type. In the other cases the type of vomiting was not stated.

In the 10 cases where it was stated that the vomiting occurred at birth, it consisted merely of regurgitation such as may be present in any normal infant. Vomiting soon after birth does not necessarily indicate congenital stenosis of the pylorus, but is to be interpreted as a hypermotility of the gastro-intestinal tract. It is our impression that in the majority of cases of congenital pyloric stenosis projectile vomiting starts during the second or third week. We believe this to be quite characteristic and an important point in the diagnosis.

Constipation: This symptom was observed in 158 cases, beginning most frequently during the third or fourth week.

Constipation:

Age	Cases.
At birth	10
First week	2
Second week	9
Third week	64
Fourth week	65
Fifth week	2
Sixth week	2
Seventh week	3
Eleventh week	1

In 63 of the cases there was no constipation, and in 1 case constipation and diarrhea were present alternately. The severity of the constipation was in direct proportion to the amount of food that went through the pylorus. In infants where 50 per cent. of the food went through the pylorus in four hours, as demonstrated by x-ray examination, there was no constipation and the stools were of a fairly good quality.

Loss of weight: In the majority of cases the loss of weight was first noticed between the second and fourth weeks. It varied from a minimum of 4 ounces to 4 pounds, 8 ounces. In most cases the loss was between 8 ounces and 1 pound.

Weight lost.	Cases.
4 ounces	4
8 ounces	31
12 ounces	33
1 pound	33
1 pound, 4 ounces	14
1 pound, 8 ounces	14
1 pound, 12 ounces	14
2 pounds	7
2 pounds, 4 ounces	7
2 pounds, 8 ounces	6
3 pounds	3
3 pounds, 4 ounces	1
4 pounds	2
4 pounds, 8 ounces	1

In 48 cases the loss of weight was unknown. In the remaining cases the weight remained stationary in 1, and in the other 2 there was a gain over a period of two months of $10\frac{1}{2}$ ounces, and 2 pounds, 7 ounces, respectively.

Peristaltic waves were observed in all cases immediately after the baby was given mother's milk from the breast or water from a nursing bottle. We have spoken of the latter as the water test. As the stomach gradually becomes filled, the typical large peristaltic waves, starting at the left hypochondrium and passing obliquely across to the right, may be seen in all cases. Usually there are two and sometimes three waves present on the abdomen at the same time.

A tumor—the hypertrophied pylorus—was definitely palpable in about 25 per cent. of the cases.

Other symptoms described in the histories of the patients studied may be listed briefly as follows:

Symptoms.	Cases.
Constant hunger	12
Restlessness	8
Irritability	2
Weakness	2
Insomnia	3
Crying	10
Anuria	5
Anuria and polyuria alternately	1
Fever (100° to 104° F.)	18
Abdominal distress after feeding	3
Coughing	5
Choking	1
Convulsions	2
Acutely ill (appearance of general collapse)	4
Icterus	3

Physical Examination.—Emaciation: Forty-three of the infants appeared to be well nourished, 125 were moderately, and 53 were extremely, emaciated.

x-Ray Examination.—The fluoroscopic examination absolutely confirms the diagnosis of pyloric stenosis made on the basis of the cardinal symptoms described above. The technic is simple. A small amount of barium is added to breast milk which the infant is given while he is being observed under a horizontal fluoroscope. The rhythmic, snake-like, peristaltic contractions seen in the pylorus, independent of the contractions of the rest of the stomach, are absolutely pathognomonic of the condition. This examination, which requires only two or

three minutes, is repeated at the end of two and four hours, at which times roentgenograms are taken. If one-half or more of the barium milk remains in the stomach at the end of four hours, the case is recognized as one of pyloric stenosis and is referred for operation. When 80 per cent. or more passes through the pylorus in four hours, although characteristic contraction waves are present, operation is deferred.

These latter cases are treated medically, and the result of treatment is frequently favorable.

It is interesting to note that in all infants more than four weeks old a large dilated stomach was noted under the fluoroscope, which was the result of a partial obstruction produced by the stenosed pylorus. The diagnosis of pyloric stenosis made under the x-ray was confirmed at operation in all cases. This comprised 177 of our patients. In 3 cases an x-ray examination had been made previous to the admission to the hospital. In 44 cases no examination was made because of the extreme emaciation and moribund condition of the infants.

In 10 cases fluoroscopic examinations were made some time after the operation. The examination showed that the stomach functioned normally in this group.

Treatment.—Preoperative treatment: As stated before, in those cases where the baby's condition is at all questionable, routine treatment is given as follows: 100 to 150 c.c. of saline are given by hypodermoclysis every four hours. Five per cent. glucose with 2 per cent. sodium bicarbonate, 1 to 2 ounces, are given per rectum every three hours. If the condition of the patient is very poor, 50 to 100 c.c. of glucose, usually followed within six hours by 60 to 80 c.c. of blood, are given through the superior longitudinal sinus.

In 12 of our cases nothing was given by mouth before the operation. In 155 cases breast milk was given in amounts varying from 2 ounces every hour to 3 ounces every three hours. In 54 cases the breast milk was supplemented with an artificial food.

The operation in most cases was performed from one to three days after the child entered the hospital.

Operation: The operation performed in all 221 cases was the usual Strauss pyloroplasty for pyloric stenosis. In 48 cases the tumor was small and no flap was made.

The condition at the time of operation was good in 63 cases, fair in 43, and poor in 115.

A general anesthetic (ether) was used in 187 cases and local anesthetic in 34 cases.

The average length of time for the operative procedure was from twelve to fifteen minutes.

At operation it was found in all cases that the tumor contracted and relaxed rhythmically just as seen under the fluoroscope. It became white, hard, and cartilage-like in character during contraction and soft and red during relaxation. The size of the tumor was found to be in direct proportion to the age of the child, or to the length of time the symptoms had been manifested. We always found a small tumor in a two or three weeks' old baby and a large tumor in a six or eight weeks' old baby. We have never found a small tumor in an older, or a large tumor in a younger, infant. The tumor usually varies from 1 to 2 cm. in diameter.

Postoperative Care.—While still on the operating table every patient was given about 150 c.c. of normal saline by hypodermoclysis. The feeding was started early, within one hour after operation. One dram of breast milk every two hours is given the first day and this is increased $\frac{1}{2}$ dram every few hours. At alternate hours water is given. Glucose is given per rectum as described above under preoperative procedure. If the infant vomits, he is started again on 1- or 2-dram feedings.

Convalescence.—There was usually fever immediately after the operation, most frequently between 100° and 101° F.

Temperature.	Cases.
98.4°– 99° F.	3
99 –100	64
100 –101	84
101 –102	19
102 –103	23
103 –104	16
104 –104.8	12

On the day the infants were discharged from the hospital the temperature was usually normal.

The pulse-rate immediately after the operation varied from 84 to 172, usually ranging from 120 to 150.

The respirations per minute were from 20 to 68, usually between 30 and 40.

Of those patients who recovered, 34 had diarrhea for the first few days and then returned to normal. In 2 cases the stools on several occasions were streaked with blood. In 187 cases the number of stools averaged from one to three daily, at first being soft and greenish, often with considerable mucus, but later becoming soft and yellow or brown.

Vomiting after operation: The infants in whom no plastic flap of the tumor was made, but the tumor simply shelled out as in the Rammstedt operation, vomited more or less for the first three days. This simpler procedure was done only in the very early cases where the tumor was small. In the other cases the more complete pyloroplasty was done, and in only 10 of the latter cases was even a slight regurgitation of the food noted during the first seventy-two hours.

In one instance the infant vomited from five to eight times daily for the first few days, and then one to three times a day for about six weeks. Following this, this infant was observed in the hospital for a month, and during this time no vomiting occurred. Another infant was readmitted to the hospital some time after operation with a diagnosis of postoperative vomiting, but was discharged improved after three days.

Condition of the Wound.—In one case where local anesthesia was used there was a large amount of purulent discharge about the wound. In 9 cases there was slight infection around the tension sutures, and in 4 cases an inflamed area around these sutures. In the remainder of the cases there was no suppuration. In 2 cases there was slight bleeding from the wound following operation, but this was immediately checked. In 10 of these 14 cases with wound complications, local anesthesia was used.

Weight curve: During the period the infants were in the hospital after operation the average gain in weight was 13

ounces.   The greatest single gain was 2 pounds, 7 ounces in twelve days.   In many cases there was a preliminary loss which was followed by a steady gain.   In 4 cases this preliminary loss was somewhat greater than the subsequent gain.   In 1 case there was a loss of 5 ounces and then maintenance of the same weight for two weeks.   All the infants continued to gain weight after leaving the hospital.   As stated above, one infant had great difficulty in becoming adjusted to any feeding formula for almost three months, over which period there was a gain of only $1\frac{1}{2}$ ounces.

In most instances the babies remained in the hospital twelve to fifteen days after the operation.

Condition at Time of Discharge.—The condition in 214 cases was improved.   The stitches had been removed, and the wound was healed in all cases.   The infants were retaining their feedings; 208 were known to be gaining in weight; 7 of the patients died.

Later History of These Infants.—Questionnaires were sent to the parents of all the infants operated on in order to learn something of the subsequent history and, if possible, to compare their development with that of normal children.   Unfortunately, however, replies were received to only 46 of the letters sent out.   A summary of the data received follows:

	Average.
Age at which infant first sat up................	$6\frac{1}{4}$ months
Age at which infant first stood.................	11 months
Age at which infant first walked................	14 months

Digestive disturbances:

Cases.	
26............................	None
1............................	Sensitive to change in diet
3............................	Occasional slight disturbances

Vomiting:

Cases.	
24............................	None
5............................	Rarely
3............................	In connection with febrile diseases

Three of the infants were somewhat constipated and 5 had poor appetites.   The general health was good in all cases.

CONCLUSIONS

1. Pyloric stenosis occurs far more frequently in male than in female infants.

2. In most cases the symptoms appear between the third and fifth weeks of life.

3. The diagnosis may be easily made on the bases of cardinal symptoms of vomiting, constipation, and loss of weight, together with the finding of visible peristaltic waves.

4. The diagnosis in all cases can be confirmed by fluoroscopic examination.

5. When the general condition of the patient is improved by giving normal saline and glucose solution preoperatively, the mortality is reduced.

6. In our hands the modified Rammstedt operation with the making of a plastic flap has given better results than the simple Rammstedt.

7. The development of the infants operated on keeps pace with that of normal infants.

8. From the statistical study of our results we continue to believe that surgical treatment, employed early in well-defined cases of pyloric stenosis, offers the safest and most certain method of relief.
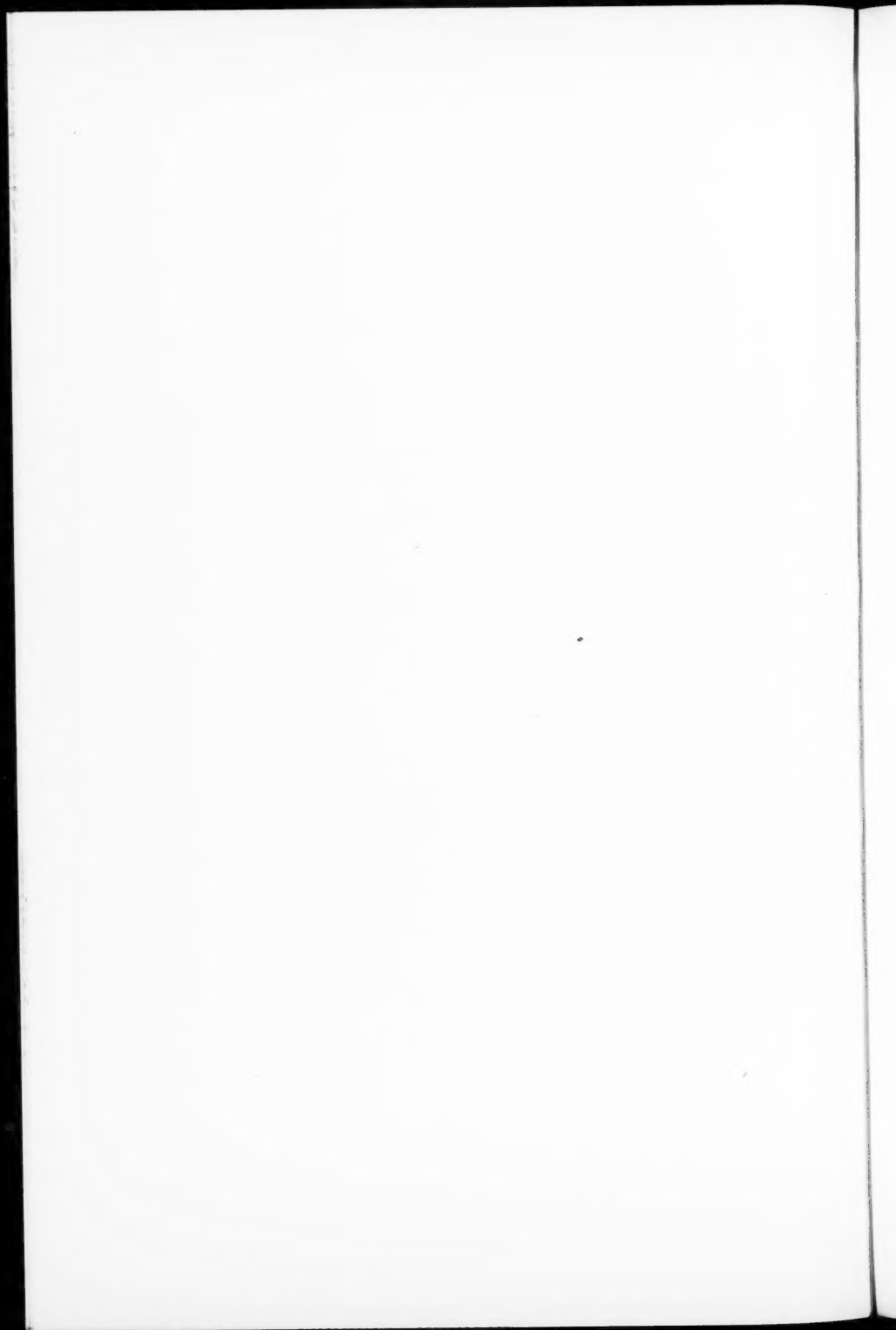
CLINIC OF DR. ARTHUR R. ELLIOTT

St. Luke's Hospital

CASE I. CEREBRAL HEMORRHAGE WITH INTENSE HYPERGLYCEMIA AND GLYCOSURIA

SATISFACTORY interpretation of the striking events in the clinical history of the case we shall now discuss is, unfortunately, not possible owing to the absence of necropsy and the development of exact anatomic data. The pathologic events represented appear to consist of hemorrhage into the midbrain with development of intense hyperglycemia and glycosuria in a woman not previously diabetic. The course of events resembles strongly those of Claude Bernard's "piqûre glycosuria" in dogs.

Hospital No. 183,414, a white, childless, married woman aged fifty-eight, admitted February 1, 1925, died February 4, 1925. No autopsy permitted. History of illness secured from patient's husband and from family physician.

According to the statement of the family physician, he had treated the patient for chronic endocervicitis, performing in November, 1924, under gas anesthesia, a curettage and removing a substance looking like partially organized blood-clot in a semi-purulent condition. No histologic examination of this material was made. Since this operation there had been a persistent slight blood-stained vaginal discharge. Urine analyses had been made several times. The last one three weeks before admission. Sugar was not present in the urine to the ordinary test.

In her preceding history there was complaint of vertigo for four or five years, dental sepsis and pyorrhea, and edema of extremities for many months. No definite history of rheumatic arthritis or endocarditis could be elicited.

Five days before admission the patient had an attack of

vomiting and purging, followed by severe pain in the right lumbar region. She was restless, stuporous, and irritable when roused, and there was incontinence of urine and feces. Twenty-four hours before entry her stupor increased and it was observed that she could not move her right arm and leg, which were, moreover, apparently tender to pressure.

Examination on admission revealed a fair state of nutrition, rectal temperature 99.6° F., respirations 20, pulse 84, blood-pressure 220/90. There was no jaundice, cyanosis, eruption or petechiæ, no adenopathy, ptosis, or exophthalmos. Many teeth were missing and those remaining showed evidence of advanced pyorrhea. The tongue was red and parched. The heart was found to be considerably enlarged and grossly fibrillating, with a systolic murmur over entire heart. Examination of the lungs proved negative except for some basal hypostasis. The liver was appreciatively enlarged. Spleen and kidneys were not palpable.

The patient was in a deep stupor and there appeared to be right-sided hemiplegia, with flaccidity and no facial asymmetry. The left pupil was larger than right, both reacting sluggishly to light. The right knee-jerk was absent, left sluggish. No Babinski toe reflex could be elicited. The entire right side appeared to be markedly tender to touch and manipulation.

The bladder was emptied of a small amount of urine by catheter. The urine was clear, specific gravity 1038, and gave an intense reaction for sugar, acetone and diacetic acid being absent. There were many hyaline casts in the urine sediment.

Further questioning of the patient's husband secured the information that for a few days preceding her present acute illness urination had increased in frequency to about every two hours during both day and night, with complaint of thirst and of transient amaurosis. Ophthalmoscopic examination was attempted, but proved unsatisfactory.

Although the patient's condition did not strongly suggest diabetic coma, she was given orange juice and glucose solution by nasal feeding tube, and 70 units of insulin were administered intravenously. These measures had no effect on her coma. As

soon as possible blood chemistry studies were made, and the report showed blood-sugar 500 mgms. per 100 c.c. of blood;
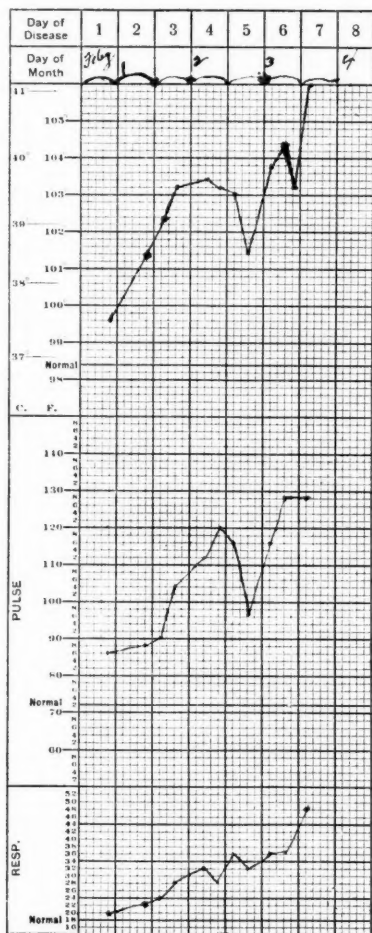
Fig. 243.

blood urea 22 mgms. per 100 c.c. of blood; total non-protein nitrogen 50 mgms. per 100 c.c. of blood.   Carbon dioxid combin-

ing power of blood 60 volume per cent. Spinal puncture yielded a clear fluid, under slight pressure, which contained 262 mgms. sugar per 100 c.c.

The body temperature during the day following admission ranged from 102.8° to 103.4° F. There was persistent convulsive twitchings of the paralyzed arm, hand, and leg, with general restlessness and marked tenderness on manipulation of the right side. Milk and lime-water were administered through nasal tube left in place, and 1000 c.c. of normal salt solution were given by hypodermoclysis. Twenty units of insulin were injected subcutaneously during the day. Blood Wassermann test and blood-culture were made, both of which were in due time reported negative. Respirations became rapid, irregular, and shallow, ranging to as high as 40 per minute. A fall of blood-pressure became apparent on the second day, reaching 150/90. Blood leukocyte count was 19,000. During the second day in hospital the temperature range gradually increased to 104.4° F., with respirations 36 to 40, irregular and shallow, without notable cyanosis. The skin was moist and cool despite high internal heat. Two urine specimens secured by catheter showed intense glycosuria, one specimen giving slight reaction for acetone bodies, while the second was acetone free. Blood withdrawn at 9 A. M. contained 995 mgms. sugar per 100 c.c., with carbon dioxid combining power of 36 volume per cent. During the day and evening 140 units of insulin were administered in divided doses, 60 units being given intravenously. The total amount of insulin administered during forty-eight hours was 260 units, of which 130 units were given intravenously. This enormous dosage with insulin resulted in a decline of blood-sugar from 995 to 405 mgm., the carbon dioxid combining power remaining practically stationary at somewhat below the normal figure. The detail of therapy and blood chemistry is shown in the chart (Fig. 244).

An antemortem rise of temperature to 106° F., with profuse diaphoresis, developed at 1 A. M., death occurring at 3.30 A. M. No autopsy was obtained.

Discussion.—Before proceeding with the discussion of the

problem presented by the interesting clinical developments in this case let us briefly review some of the facts in the pathologic physiology of glycemia.

The concentration of sugar in the blood of the systemic circulation is maintained in health at a tolerably constant level ranging from 0.080 to 0.128 per cent., representing the reciprocal relationship between production and loss by oxidation.   The

Transcript of blood chemistry and insulin dosage

Time	B. P.	Blood Urea	N. P. N.	Spinal Fluid Sugar	Blood Sugar	Co₂ Combining Power	Insulin
2/1/25 4 P. M.	222/90						30 units intravenous
5:45 P. M.							40 units
10: P. M.							10 units
2/2/25 1 A. M.							10 units
7 A. M.		22	50		500	60	10 units
10 A. M.	160/90			262			
2/3/25 9 A. M.					995	36	
12:30 P. M.							20 units
4 P. M.	160/90				930		20 units
6:45 P.M.							20 units
8:30 P. M.	134/90				770	52.6	60 units intravenous
9:30 P. M.	98/70						
10:15 P. M.	94/74				650	42.8	
2/4/25 12:30 A. M.	84/70				408	42.5	40 units
							Total 260 units

Fig. 244.

chief sources of supply for blood-sugar are the liver and, to a much less extent, the muscles.   In these tissues the sugar derived from food intake is stored away as glycogen, which can be reconverted into sugar and delivered again to the blood whenever required to maintain the blood quota constant.   The formation of sugar from glycogen has been termed "glycogenolysis."   Discharge of sugar from the liver in amounts above physiologic requirement is no doubt the commonest cause of

most forms of hyperglycemia, at least of those that are transitory. It appears to be a fact that glycogenolysis in the liver and resulting sugar concentration in the blood is subject to control from the nerve centers. The occurrence of glycosuria as a symptom in certain cases of brain tumors and brain injuries and its association with certain neurotic conditions appear to bear upon this relationship. Definite localization of a diabetic center in the brain is so far not established, but experimental methods indicate regulation of glycogenolysis from some portion of the midbrain between the cerebrum and the cord. Efferent impulses proceeding from this glycemic area cause discharge of glycogen from the liver by stimulation via the splanchnic nerves. The classic experimental example of glycosuria produced by central stimulation is the so-called "piqûre glycosuria" made familiar by the work of Claude Bernard. In the dog and rabbit puncture of the floor of the fourth ventricle is invariably followed by hyperglycemia and glycosuria provided the liver contains glycogen. Whether these results of piqûre are really due to direct nerve stimulation of glycogenolysis in the liver cells or may not be secondary to some other factor which brings about the same effect, such as adrenal stimulation or morbid vasodilation in the liver, remains still a matter of controversy. If, previous to the experiment, removal of the liver from the circulation has been secured by anastomosis of the portal vein with the vena cava, the glycosuria which is produced in the normal animal by piqûre does not occur. It seems a natural conclusion, therefore, that piqûre stimulates the glycogenolytic process in the liver, and that it fails to set going a similar effect in the muscles where there are present also considerable stores of glycogen.

Analysis of the Case.—We must first consider whether the patient was, previous to her last illness, already a diabetic who developed hemiplegia with aggravation of a pre-existing hyperglycemia and glycosuria, or whether a hemorrhage into the midbrain involving the region of the fourth ventricle induced a "piqûre glycosuria" similar in type to experimental glycosuria from puncture of the floor of the fourth ventricle in dogs. Against the former interpretation may be ranged as evidence the ex-

cellent state of her nutrition, absence of glycosuria to within three weeks (date of latest urine analysis) of final illness, absence of ketosis, and the peculiar course of the hyperglycemia under observation. The patient was evidently an old hypertensive cardiorenal case with fibrillating sclerotic heart, and her urinary frequency preceding final illness might be accounted for on that basis. With the onset of cerebral symptoms urination became hourly and thirst excessive, thereby signaling the onset of glycogenolysis. The cerebral hemorrhage was evidently basal in location, judging from disturbance of respiration, athetoid convulsive movements, and sensory involvement. The intense and increasing hyperglycemia little influenced by massive insulin administration appears to indicate its central origin. In Bernard's "piqûre glycosuria" glycogenolysis in the liver under central nervous stimulation proceeds with extreme rapidity until the liver is completely emptied of glycogen. In dogs this is ordinarily accomplished within forty-eight hours, the blood-sugar and urine by this time returning to normal. There is no ketosis. This is in notable contrast to experimental pancreatic diabetes which persists after depletion of glycogen because of formation of sugar from body proteins (neoglycogenolysis) with secondary ketosis. When we study the phenomena of our case it seems clear that we are dealing with a central nervous glycosuria. The hyperglycemia was fulminating in its intensity, resistant to oxidative therapy, unaccompanied by ketosis, and had the patient lived long enough we might have witnessed spontaneous disappearance of the hyperglycemia and glycosuria when the liver glycogen had become exhausted. Unfortunately, this case, following the rule where there is glycosuria resulting from cerebral apoplexy, proved fatal too soon to determine whether the glycosuria would end spontaneously or develop into a true diabetes. Cases of this type have usually displayed at necropsy hemorrhage into the region of the fourth ventricle. The glycosuria is usually light, averaging 8 to 10 to 12 grams per liter. In 2 cases reported by Lepine it reached a concentration of 25 and 40 grams per liter. I am not in possession of any records of blood-sugar analyses in similar cases. That it is not a frequent development

in cerebral apoplexy seems apparent from common experience. One observer tested the urine of 50 cases of recent hemiplegia without finding it in a single case.

Fractures of the skull, especially basal fractures, may be followed by glycosuria, and it may develop as a very transient phenomenon after cerebral concussion.

CASE II. TUBERCULOUS LOBAR PNEUMONIA

INFLUENCED by our experience with pulmonary tuberculosis as it is ordinarily encountered in practice and in the clinics and hospital wards, we are apt to regard the disease as a chronic one. This undoubtedly holds true for the majority of cases, but we must not lose sight of the fact that it may occur in the acute form.

In contrast to the usual chronic type these infrequent cases appear to develop suddenly and run a rapid febrile course to fatal issue within a few weeks. If the patient be a child, the disease may terminate life quickly. This may also hold true for adults, although among the mature the progress of the disease is apt to be more deliberate, and may eventually lose its acute characteristics and become in time subchronic or chronic.

Various names have been employed to designate this relatively small group of acute pulmonary tuberculosis. A few of these names are acute pneumonic phthisis, phthisis florida, galloping consumption, and tuberculous pneumonia. Most cases are recognized clinically as bronchopneumonia. This is its characteristic development when it occurs among infants and young children. This is true also in the negro, even among adults of that race, although much more rare in the white adult. When the disease is encountered involving an entire lobe or the whole of one lung, and appears to have had a sudden onset with high fever, pleural pain, rusty sputum, and with the physical signs of lung consolidation, its resemblance to lobar pneumonia is so great as to lead to the utmost difficulty in differentiation. This form of the disease has been called "tuberculous lobar pneumonia." The following briefly described instance will furnish us material for discussion of the problems surrounding its recognition.

Hospital No. 171,113. Negress, single, aged seventeen, admitted September 7, 1923. Died October 28, 1923. Admittance diagnosis, lobar pneumonia; discharge diagnosis, tuberculous

lobar pneumonia, acute pneumonic phthisis, miliary tuberculosis. Autopsy No. 58, series of 1923.

Patient's past medical history, so far as it could be secured, is entirely devoid of significant illness. Family history, no data procurable.

Present illness began four weeks before admittance with symptoms of a nasopharyngeal cold and stiff neck. Cough began within a few days and her body temperature was found to be 103° F. Expectoration was moderately free, yellowish in color, and on one occasion frankly blood tinged. Pain and stiffness on movement in the front and sides of the neck persisted for three weeks and then disappeared. One week before entry her condition grew suddenly worse, with the development of severe pleural pains in the left thoracic area and pressure soreness over the front and lateral aspects of right chest. Fever ranged around 103° F. Cough distressing and sputum scanty.

On admittance temperature was 104.2° F., pulse 130, respirations 30, blood-pressure 100/60, nutrition poor, body weight about 90 pounds. Blood-count, erythrocytes, 4,640,000, leukocytes 9150. Widal test negative. Blood Wassermann negative. Sputum, tubercle bacilli absent, numerous leukocytes, Gram stain, numerous Gram-positive cocci in chains, numerous fusiform bacilli and spirilli, numerous Gram-negative cocci in clusters (Micrococcus catarrhalis), blood-culture (reported September 19th) no growth of pathogenic bacteria after seven days' incubation.

Physical examination—left chest: Expansion and excursion limited. Marked percussion dulness, with increased resistance over entire left lung. Bronchial breathing over upper lobe, diminished breath sounds over lower lobe, vocal fremitus increased over upper lobe, absent over lower lobe. Many moist and crepitant râles on deep breathing over entire left lung. No pleural friction detected.

Right chest, expansion good. Percussion and breath sounds normal.

Heart normal in size and position. No murmur or pericardial friction. Abdomen negative.

x-Ray plates of the chest (Fig. 245) reveals dense shadow involving entire left lung, causing complete obliteration of lung transparency. The heart shadow shows no displacement to the right. The right lung shows some peribronchial infiltration, with a small patch of what appears to be consolidation in the lower part of right upper lobe.
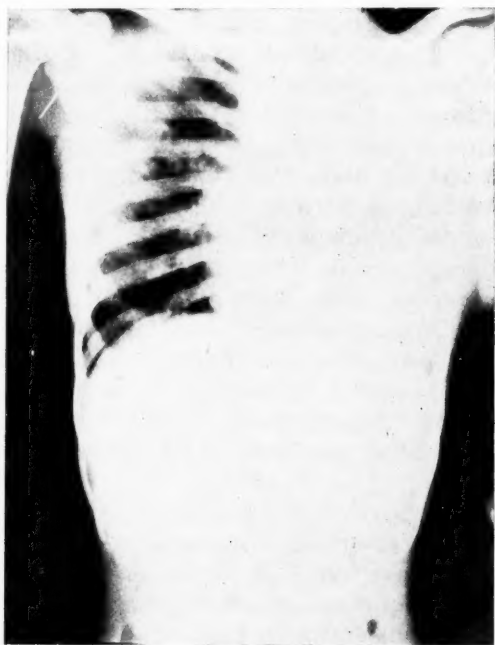
Fig. 245.

Both physical findings and *x*-ray evidence pointed to massive consolidation of the left lung, with possibly some pleural exudate. The chest was aspirated and 25 c.c. of cloudy, straw-colored, and slightly blood-tinged fluid withdrawn. This proved negative to culture after forty-eight hours' incubation. Direct stain showed many leukocytes, but no bacteria, and acid-fast stain disclosed no acid-fast bacilli. Cell count 1922 per c.mm., differential

count, lymphocytes 90 per cent, polymorphonuclears 10 per cent.

Tentative diagnosis: Massive tuberculous pneumonia, miliary tuberculosis.

The patient's condition remained unchanged for several days, temperature varying between 102.6° and 104.2° F., with weak, rapid pulse, and no change in the character of the physical findings. A second attempt at paracentesis was disappointing, no fluid being obtained.

Progress note September 29th, three weeks after entrance, states that there is no change, the patient's condition remaining in general about the same except for growing weakness and frequent complaint of pain in the left chest. Temperature range reached a maximum of 105° F., pulse from 116 to 134. Pressure sores developed and several pustules the size of a dime appeared on the patellar surfaces. Chest examination showed complete involvement of the left lung without any very definite findings in the right chest. A second x-ray plate failed to reveal any additional findings. Blood examination, erythrocytes 4,680,000, leukocytes 8450. The urine contained a plain trace of albumin and a few casts. The left chest was needled for the third time, without securing any fluid. Sputum remains free from acid-fast bacilli.

During the remainder of the clinical course until death from exhaustion October 28th, fifty-one days after entry, there was no important change in physical signs. Blood examinations revealed a steadily progressive anemia and leukopenia of from 5500 on October 10th to 3350 on October 27th. All sputum examinations were reported negative for tubercle bacilli. Multiple bed-sores and skin abscesses developed.

A striking feature of the case was the sustained high temperature, daily maximum never being less than 103° F. and as high as 105° F., while minimum temperature never fell below 101° F.

Excerpts from Autopsy.—There are about 100 c.cm. of clear, straw-colored fluid in the pericardial sac. The vessels of the pericardium are slightly prominent. The right pleural cavity

contains about 150 c.c. of a clear, straw-colored fluid. The left pleural cavity is entirely obliterated by easily torn fibrous adhesions. The left lung has a consistency that approaches that of cartilage.

The lining of the inferior vena cava is bluish gray and smooth. One cm. above its bifurcation is a white thrombus which extends into both common iliac veins, and from there it continues as a grayish-red thrombus into the left external iliac, and as a gray thrombus into the left internal iliac, and also into the right external iliac and the right internal iliac veins. These are traced as far as the inguinal ligament in the external iliac, but were not traced any further, and the thrombi are also traced into the smaller branches of the internal iliac veins. They are attached to the walls of the vessels and apparently entirely occlude the lumen of the vessels. The lining of the aorta is yellowish white and smooth. The tracheobronchial lymph-glands are pigmented with coal-dust, but are otherwise normal. The lining of the esophagus is grayish red and normally rugated. The trachea and main bronchi contain frothy mucus; their lining is grayish pink and unchanged. At the region of the thymus are four enlarged lymph-glands, the largest 4 cm. long, with firm, cartilage-like nodules which contain no caseation or calcification. There is moderate edema of the mediastinal tissues.

The heart, with 6 cm. of attached aorta, weighs 120 grams, and is the size of the owner's fist. The epicardial tissues are smooth and glistening everywhere. The lining of the heart chambers and the covering of the valves are everywhere smooth and glistening. The mitral valve admits the end joints of two fingers, the tricuspid three. The lining of the root of the aorta is lemon yellow and smooth everywhere. The lining of the coronary arteries is yellow and smooth, their lumens are of normal size. The myocardium has a maximum thickness in the wall of the left ventricle of 10 mm., is reddish brown throughout, with no areas of fibrous tissue. The foramen ovale is closed. There is no change in the lining of the coronary sinus. The left lung weighs 900 grams. It is grayish yellow for about three-fourths of its entire surface, except for small areas which are grayish

black, with coal-dust and a few small red areas separating the grayish-yellow regions in the dorsal region of the lung. Practically the entire lung is covered by a fibrin coating which is thickest on the diaphragmatic surface, where it has a maximum thickness of 2.5 mm. On broad surfaces made by sectioning the lung a cavity is exposed in the upper lobe close to the interlobar fissure and 1 cm. from the dorsal surface of the lung, which is 2.5 cm. in diameter and filled with creamy caseous substance. The lining of the cavity is only about 1 mm. thick, is dirty gray, and contains many small grayish-yellow tubercles. Around the cavity in all directions for 0.5 to 2.5 cm. is red lung tissue which,

Fig. 246.

however, is not normally crepitant, and the remaining lung tissue is grayish yellow except for very few small red areas corresponding to those described on the surface. The lining of the bronchi, large and small, is grayish pink and covered with a yellowish-gray gelatinous and caseous material. The lining of the branches of the pulmonary artery and veins is smooth and unchanged. In all, about 95 per cent. of the lung is solid. The right lung weighs 250 grams, is grayish pink outside, mottled grayish black, about one-half by coal-dust. The surface everywhere is studded with small gray slightly raised tubercles, averaging 1 mm. in diameter. A few of these are in the pleura or close to it, but most

are in the lung substance proper. The lung substance is crepitant throughout except for these small areas, which are easily palpable, the largest of which is 3 mm. in diameter. Broad surfaces made by sectioning the lung are grayish red, mottled about one-third in the dorsal region by the tubercles which are firm and round. There is one place in the dorsal part of the lower lobe about 1.5 cm. in diameter, which is firmer than the rest of the lung tissue, due to the conglomeration of many tubercles, with some infiltration of the surrounding tissue. The lining of the small divisions of the bronchi is grayish pink and smooth, and there is very little gray mucus. The lining of the small branches of the pulmonary artery and veins is grossly unchanged.

The spleen weighs 100 grams, is reddish purple outside with a normal wrinkled capsule. There are about twenty-five small round grayish-yellow bodies seen beneath the capsule toward the convex surface, the largest of which is 1.5 mm. in diameter. Surfaces made by sectioning the spleen are reddish brown and normally firm, the fibrous septa plainly visible, and here and there are small grayish-yellow spheric bodies, resembling tubercles, not closer together than 1 to 3 cm. The adrenals are almost devoid of yellow lipoid material and are of normal size and shape. The right kidney is of normal size and grayish red. The capsule strips easily, leaving a smooth surface which is somewhat gray. The red stellate veins are clearly visible. The cortex is generally 8.5 mm. thick, the markings rather indistinct, and in places the pyramids are not clearly demarcated. There are a few spheric gray to grayish-red bodies, not over 1.5 mm. in diameter, in the cortex of each kidney, but not nearly as numerous as in the spleen or liver. The edges of the kidney, after cutting, roll out slightly, and the kidney substance on surfaces made by sectioning is pale, with small red punctate areas, as from hemorrhage, and there are a few irregular grayish-yellow areas in the cortex 2 to 3 mm. in diameter, which are not as hard as are the tubercles. The left kidney is like the right in all important respects, and together they weigh 290 grams. The liver weighs 1020 grams, is reddish brown, mottled about one-fifth with regular grayish-yellow to yellowish-brown areas from

1 to 15 mm. in diameter, and these places are not hard. There are also widely distributed over the surface of the liver grayish-white, more translucent spheric bodies, 1 to 2 mm. in diameter, not involving more than one-fiftieth of the surface of the liver. The front edge of the liver is sharp. Broad surfaces made by sectioning are reddish brown and essentially like the outer surface, mottled with irregular yellowish-brown areas and the fewer gray translucent tubercles, and the liver substance in general has an opaque, cloudy appearance. The gall-bladder contains about 10 c.c. of clear, viscid golden-yellow bile, and its lining, as is the lining of the cystic duct, is unchanged, except for bile staining. The lining of the hepatic artery is smooth and unchanged. The pancreas is grayish pink inside and outside and is normally lobulated. The intestine contains a small amount of yellowish-green feces, and its lining is unchanged except for a few petechial hemorrhages in the middle portion of the ileum in one of the dependent loops. There are about 200 c.c. of thin dark brown fluid in the stomach having the characteristic sour odor. The lining is normally rugated and contains a few petechial hemorrhages along the greater curvature.

There is no change of the deep scalp tissues. The calvarium comes away easily. The top half of the outside of the dura is gray and smooth. Nothing is in the superior longitudinal sinus but loosely clotted dark red blood in its posterior third. The inside of the top half of the dura is grayish blue and glistening. The cerebrospinal fluid is clear. The leptomeninges are transparent and there is no edema of them or any other noteworthy change. The cerebral convolutions are well rounded and the arteries at the base of the brain are normally thin walled. The inside of the bottom half of the dura is bluish gray and glistening and its sinuses are empty. The inside of the bottom half of the cranium is grayish-yellow intact bone. The middle-ear cavities are normal, their linings and the drum membrane are normal. The sphenoid, ethmoid, and frontal sinuses are normal and their lining unchanged.

Pus from Right Axilla.—Direct stain (Gram): Many leukocytes; large Gram-positive cocci in pairs and tetrads. Cultures:

Gram-positive cocci in tetrads and clusters (Staphylococcus albus).

Pus from Left Axilla.—Direct stain (Gram): Many Gram-positive cocci in pairs; many leukocytes. Cultures: Gram-positive cocci in pairs and clusters (Staphylococcus albus); Gram-positive bacilli (hay bacilli).

Histology.—Lungs: There are extensive regions of caseation necrosis in the lung tissue and the alveolar spaces contain many mononuclear cells and fibrin, cellular reactions like those occurring with an extensive pulmonary tuberculosis. The destruction of tissues is marked and, inasmuch as acid-fast bacilli have been demonstrated in the fresh tissues, an examination for these bacteria is not made at the present time. Spleen: There are many miliary tubercles forming regions of necrosis up to 1 mm. in size. There are similar tubercles in the liver and lymph-glands.

Pathologic diagnosis: Tuberculous lobar pneumonia of entire left lung; generalized miliary tuberculosis of the viscera; left fibrinous pleuritis.

Discussion.—The medical history of this patient previous to coming under observation is, unfortunately, very meager, and furnishes us no hint as to the nature of the primary infection. After a preliminary illness extending over approximately a fortnight, characterized by sore throat, stiff neck, and irregular fever, she became rather suddenly incapacitated with chest pains, high fever, and cough. There was no chill or rigor to usher in this sudden change. One of the important things in the diagnosis of acute pneumonic phthisis is a good account of the patient's previous health. Unfortunately, such was not forthcoming in this case. When details are available it may appear that preceding the advent of fever and acute pulmonary symptoms the patient has been in unsatisfactory health, has had a chronic cough, a previous hemoptysis or night-sweats, or has been losing slowly in weight for some time. Prodromal symptoms of this suggestive character are of great diagnostic importance, as they raise a strong presumption of pre-existing incipient or chronic tuberculosis. Without aid from this source

one is forced to depend on clinical developments and such proof as is available from sputum examinations and *x*-ray findings to determine the true nature of the pneumonia. Clinical criteria are, unfortunately, not very reliable. Onset may be abrupt, with premonitory chill, early chest pains, and high temperature. It is true that pain in the chest is usually milder in tuberculous than in non-tuberculous pneumonia and dyspnea less marked from the beginning, but these variations will not establish the diagnosis, nor will the fact that cough is earlier and more annoying in tubercular pneumonia. In both forms of pneumonia the sputum is usually blood-tinged or rusty. A frank hemoptysis occurring early after onset should always awaken suspicion that we are dealing with tuberculosis. Should softening and caseation set in early, the sputum may contain large numbers of tubercle bacilli and the diagnosis become obvious, but ordinarily in the beginning and even throughout the entire duration of the case repeated staining of the sputum may fail to reveal any acid-fast bacilli, and because, on the contrary, pneumococci are usually present in numbers the diagnosis is difficult. The case just recited furnishes a striking instance in point, for although repeated careful examination of the sputum was made we were never able to demonstrate their presence. This was the more surprising considering the fact that a large area of softening was found at autopsy to exist in the left upper lobe.

Careful study of the temperature chart may furnish valuable information. Perhaps more frequently than by any other means the behavior of the patient's fever awakens our suspicion as to the true situation. During the first week the temperature curve is of a continuous type like true lobar pneumonia, and may range equally high. During the second week, when ordinarily in pneumonia we expect abrupt critical decline to normal or at least defervescence, nothing of the kind occurs, fever continues unabated, with a decided tendency to become hectic. Morning remissions may decline to normal or below, the afternoon rise attaining 103° F. or higher, with copious sweats, exhaustion, and low blood-pressure. To the attentive observer such a tem-

perature needs no interpretation. There is, however, no rule in the matter of temperatures in pneumonia of any type. Anomalies of temperature are familiar facts. And this is true as well in tuberculous pneumonia. In the case we have been studying the fever was high and sustained throughout, daily maximum never being recorded lower than 103° F. and daily minimum never less than 101? F. After all, the suggestive thing about this temperature curve in tuberculous pneumonia is its quantitative rather than its qualitative aspects. Its continuance beyond the period alloted by experience to non-tuberculous pneumonia should awaken suspicion not of "unresolved pneumonia," that figment of an uninformed imagination, but of tuberculous pneumonia. It is at this point that we should again search the sputum carefully for tubercle bacilli, and they will in the majority of cases be found. x-Ray plates of the chest may aid considerably in the diagnosis of doubtful cases. Evidence of cavity formation in areas not too densely consolidated or signs of tuberculous infiltration in the opposite lung will at once awaken suspicion.

In recapitulation the following points may be emphasized: Tuberculous lobar pneumonia, although a rare disease, may occur in anyone's experience, and its close resemblance both in clinical development and physical signs to acute lobar pneumonia of non-tuberculous origin may lead to error in diagnosis, especially during the earlier stages. Prognosis is bad, consequently it is of great importance to make the differentiation as early as possible. A positive sputum may accomplish this, but, lacking evidence of this kind, one may have to wait for the case to develop before suspicion becomes aroused as to the true nature of the process. The clinical criteria available for differentiation are evidence secured by x-ray examination, prolonged sustained temperature tending to become hectic in type, marked asthenia, rapid and extreme emaciation, pulmonary hemorrhages, copious sweats, rapid small pulse, and low blood-pressure. A careful and detailed medical history may prove of the greatest importance in diagnosis.
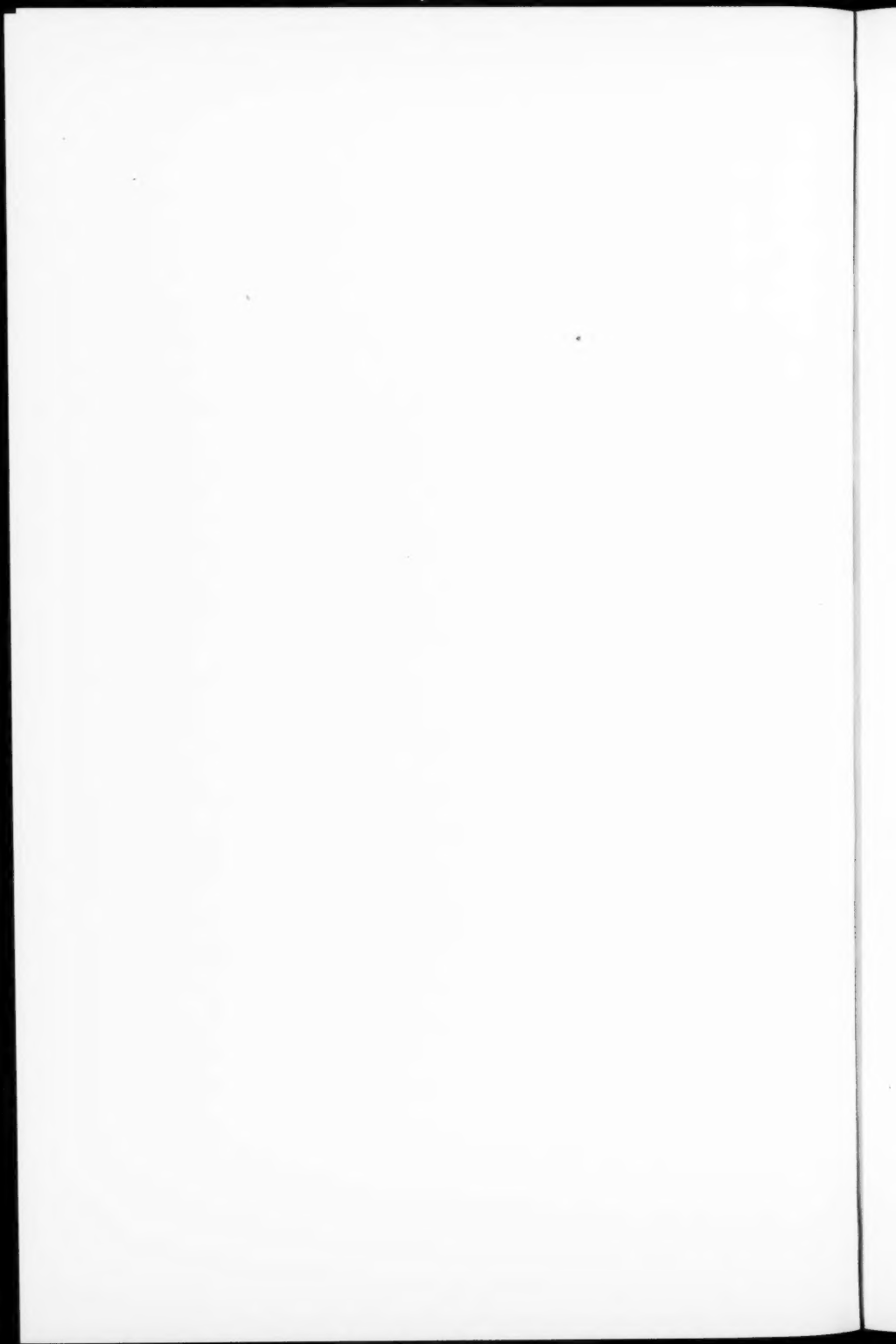
CLINIC OF DR. WALTER S. PRIEST

MICHAEL REESE AND CHICAGO MEMORIAL HOSPITALS

CASES OF HYPERTHYROIDISM SIMULATING PRIMARY HEART DISEASE

CARDIAC disturbances associated with frank exophthalmic goiter have long been recognized, and the diagnosis of the underlying condition presents no difficulties. Comparatively few, however, have called attention to a type of hyperthyroidism without goiter or exophthalmos in which the chief complaints and physical findings seem purely cardiac in origin. Boothby, in Oxford Medicine, Vol. III, gives a good description of this type. Levine and Sturgis[1] have recently called attention to it in a report of 5 cases, and Dameshek[2] mentions it in a study of the heart in hyperthyroidism.

In 2 of the following cases the real source of trouble was apparently overlooked for some time. In the third the importance of a goiter which had existed for some time was underestimated.

Case I.—An unmarried woman, age fifty-three, referred by Dr. R. B. Bettman, was first seen December 3, 1924. Her complaints were leakage of the heart, palpitation, dyspnea on mild exertion, nervousness, and slight edema of the ankles in the afternoon. Three years previous she had had a mild attack of influenza from which recovery was unusually slow. At that time she first noticed palpitation and her physician told her she had leakage of the heart. After several weeks she was able to resume her occupation as a seamstress. About a year following the influenza she had another attack of "heart trouble" characterized by palpitation, some dyspnea, and weakness. In

August, 1924 the palpitation again became troublesome, but she did not consult a physician until the latter part of October, at which time she was told she had high blood-pressure and heart trouble. She was kept in bed three and a half weeks, given digitalis, and instructed to watch her pulse-rate which at first ranged from 120 to 140. When the pulse-rate reached 92 she was permitted to get out of bed. A significant observation made by the patient was that the presence of visitors, including her physician whom she had known for years, would cause her pulse-rate to increase from around 92 to 120 or 130. During the two and a half weeks following her stay in bed she felt little if any better, the palpitation was still present, and she noticed her ankles swelling a little. Two points in the past history are significant. As a child she was supposed to have had a weak heart, and fifteen years prior to the present illness she had been told that her heart was jerky and irregular. Menopause had occurred uneventfully three years previous. Her appetite was good, bowels normal, sleep undisturbed. She had recently lost some weight. Other than the influenza no acute infectious diseases had occurred. She had never been aware of any enlargement of the neck.

With such a history one might well suspect organic heart disease with recurring attacks of decompensation.

On examination, the pulse was 144, almost Corrigan in type, but regular. The tachycardia persisted after resting in the recumbent position for twenty minutes. She was extremely nervous and had an anxious, startled expression out of proportion to the apparent acuteness of her illness. Marked vasomotor instability, as evidenced by alternate blanching and flushing of the skin, was present. The face was flushed. Temperature was normal. Blood-pressure was 192/90. Respirations were 30 per minute and not increased on lying down.

No cyanosis was present. The heart was slightly enlarged, being 13.5 cm. to the left and 3.5 cm. to the right in the fifth interspace. A loud blowing systolic murmur was audible over the entire precordium, with maximum intensity at the apex. Transmission was poor. The second apical tone was faint and

the second pulmonic accentuated.   The cardiac impulse was forceful, diffuse, and heaving.   The vessels of the neck were markedly engorged and pulsating.   The thyroid appeared somewhat enlarged, although this was made more apparent by the enlarged vessels.   It was not such as to attract attention on casual observation.   There was no exophthalmos and no lid signs were present.   The thyroid did not feel nodular.   There was slight tremor of the fingers and perspiration was excessive. The lungs were clear throughout.   The liver was not enlarged or tender.   No hemorrhoids were present.   The ankles pitted slightly on pressure.

A tentative diagnosis of adenoma with hyperthyroidism was made, and the patient referred to the Michael Reese Hospital for further study.

After six days of bed rest, during which time the pulse-rate ranged from 100 to 140, the temperature from 98° to 99.6° F., and respirations from 24 to 28, the basal metabolism was $+60$ per cent.   The following day Lugol's solution, 10 minims three times a day, was started.   On this day she had an attack of paroxysmal tachycardia lasting about three hours, the pulse going to 160.   Following this the pulse-rate dropped rapidly and the fifth day after starting Lugol's solution the average rate was 88.   Temperature and respirations were normal, and considerable improvement in subjective symptoms was noted. Three days later another attack of paroxysmal tachycardia occurred, and another two days after this.   Metabolism at this time was $+47.8$ per cent.   It was feared that the optimum time for surgical intervention had passed, but two days later the pulse had dropped to 80, and, under ethylene anesthesia, Dr. Bettman performed a subtotal thyroidectomy.   Fully half of the gland was substernal, accounting in a measure for the lack of noticeable enlargement.

Postoperative course was uneventful.   Lugol's solution, 5 minims three times a day, was given for a few days, but stopped because of nausea.   She left the hospital on the nineteenth postoperative day, at which time the pulse averaged 88.   The nervous manifestations were gone, tremor absent, metabolism $+5.6$ per

cent., blood-pressure 160/80, urine (which had contained a trace of albumin) normal. The heart murmur was scarcely audible.

An electrocardiogram taken on admission (Fig. 247, *A*) showed nothing unusual aside from muscular tremor. A distance heart plate showed a transverse measurement of 13 cm. and a chest measurement of 30.5 cm. (slightly enlarged). Blood nitrogen was a little high (N. P. N. 48 mg. per 100 c.c.). Sugar was normal (82 mg. per 100 c.c.).

For six months after leaving the hospital the patient did very well, worked daily, was free from her former symptoms, and gained weight. In July, 1925, following an emotional upset, the palpitation, tachycardia, and dyspnea returned. In addition, precordial pain was present at times. Examination showed a pulse-rate of 164, blood-pressure 180/80, and other findings as before.

At first the inclination was to explain the symptoms and findings on the basis of a damaged heart, but as no improvement followed three weeks' rest in bed and digitalis she was again referred to the hospital. Metabolism was +62 per cent. During the period of observation she had repeated attacks of paroxysmal tachycardia during one of which an electrocardiogram was taken (Fig. 247, *B*). One of these attacks was associated with mild hallucination, and all with precordial pain. Lugol's solution did not produce the striking effect noted at the first admission, although an average pulse-rate of 94 was finally obtained. The general condition did not make her a good operative risk, and x-ray therapy was tried. The technic as described in Case II was used, except that the dosage was 300 Roentgen units per field. No improvement could be noticed after three treatments. During this time a definitely palpable mass had developed on the left side of the neck. Apparently we were dealing with a recurrent adenoma which would not respond to medical or x-ray therapy. The heart was not increased in size over that at the first admission. The lungs were clear, liver not enlarged, and no edema was present.

Under gas-oxygen-ether anesthesia Dr. Bettman removed the gland on the left side which was almost as large as the orig-
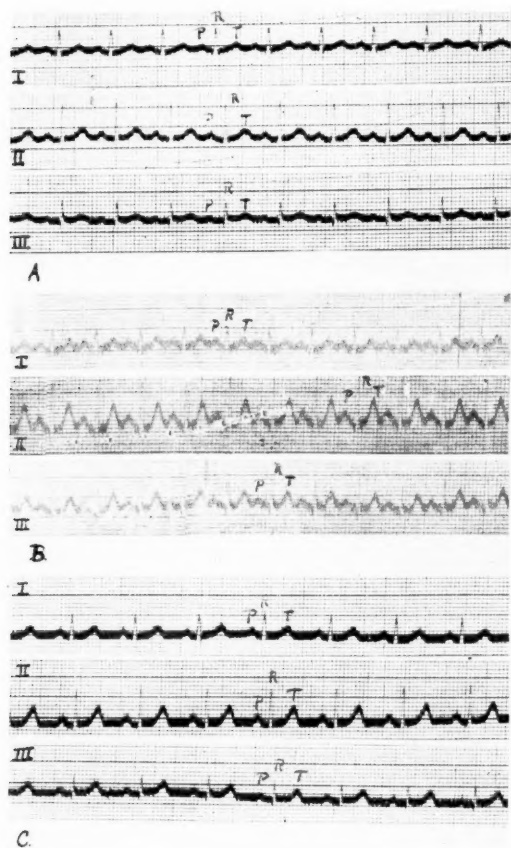
Fig. 247.—Case 1. *A*, Tracing taken December 4, 1924. Normal mechanism. Muscular tremor. *B*, Tracing taken August 12, 1925, during an attack of paroxysmal tachycardia of auricular origin. Rate 150. Abnormally high P and T waves, especially in Lead II. *C*, Tracing taken August 25, 1925, twelve days after operation. Normal mechanism. Rate 90. Decrease in amplitude of P and T deflections.

inal.   Because of her rather poor condition the right side was not explored.   Postoperative course was again uneventful, the pulse-rate dropping rapidly to 88, where it remained until discharge

on the seventeenth postoperative day. Metabolism at this time
was +13 per cent. and blood-pressure 120/80. The improve-
ment in subjective symptoms was striking. The heart murmur
had entirely disappeared. At present the patient is still under
close observation. She is gaining in weight and the pulse re-
mains around 88. Subjective improvement has been continuous.

Case II.—Male, age thirty-nine. Metallurgist. First seen
August 14, 1924. Complained of palpitation, nervousness, dysp-
nea on exertion, and generalized aching pains. Symptoms had
been present for ten days. About one year previous, on getting
out of bed one night, he had experienced an attack of palpita-
tion followed by a fainting spell. At the time he had been work-
ing unusually hard. His physician told him he had a "floppy
heart," and advised rest. About one month later another at-
tack of palpitation occurred. From that time on he felt below
par, was irritable, worried considerably about himself and busi-
ness affairs, and was subject to fits of depression. He had had
typhoid at twenty-four, influenza in 1919 and 1923, and an ap-
pendectomy in 1920.

On examination the resting pulse-rate was 90, temperature
normal, respirations 20, blood-pressure 112/60. The tonsils ap-
peared ragged, but no pus could be expressed. There were a
few crowned teeth and old roots. The thyroid was slightly and
diffusely enlarged, but not enough to have called his attention to
it. No nodule could be felt. The neck measured 37 cm. in cir-
cumference. No unusual pulsation of the vessels was present
and exophthalmos and lid signs were absent. Excess sweating
and slight tremor of the hands were present. The general de-
meanor was that of an easily excited, nervous individual con-
siderably worried about himself. The deep tendon reflexes were
equal, but markedly exaggerated. Urine, blood-counts, and hemo-
globin were normal. Differential showed a slight increase in
mononuclear cells, a finding suggestive of hyperthyroidism ac-
cording to some observers. Blood Wassermann was negative.
Because of close contact with lead in his profession careful search
was made for signs of chronic plumbism, but none found. The

heart was not enlarged either to percussion or x-ray and there were no murmurs. The apex impulse was more heaving and forceful than normal. Marked sinus arhythmia was present, an unusual finding in an adult of his age (Fig. 248). P and T of the electrocardiogram were unusually high in Lead II. Aside from an easily reducible inguinal hernia the remainder of the physical findings were normal.

The evidence for hyperthyroidism was certainly not great. The findings were more characteristic of that rather indefinite group of cases called "neurocirculatory asthenia." But a few days later he was observed during an attack of palpitation, at
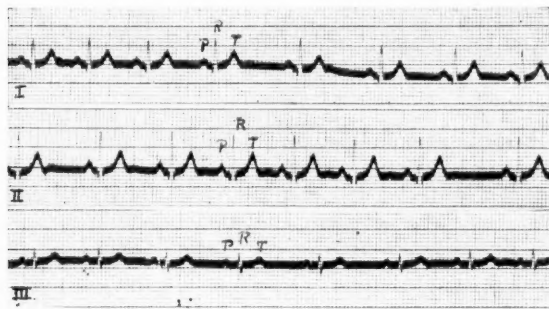
Fig. 248.—Case II. Tracing taken August 21, 1924. Sinus arhythmia. Rate approximately 84. P_2 and T_2 unusually high.

which time the pulse-rate varied from 130 to 140. The attack came on suddenly and disappeared suddenly while under observation. A definitely more marked pulsation of the thyroid vessels was noted at this time. Accordingly, a metabolic rate determination was made and found to be +35 per cent. The accuracy of the reading was checked by a second determination.

A diagnosis of mild or early hyperthyroidism was made. He was told that operation was the treatment of choice, but because of the apparent mildness of the condition and lack of evidence of cardiac damage it would be safe to try x-ray therapy first. He elected the latter, and was referred to the Roentgenological Department of Michael Reese Hospital, where treatment was

carried out under direction of Dr. H. M. Beets, to whom I am indebted for the following data:

Five exposures were given, one every fifth day, the first over the right lobe, the second over the left lobe, and the remaining three anteriorly. The rays were generated by a potential of 200,000 volts, the tube current was 3 milliamperes, and the rays filtered through copper 0.25 mm. thick. The target patient distance was 50 cm., the time of treatment fifteen minutes each. The dosage to each field was 150 Roentgen or electrostatic units. The size of the fields was 14 by 14 cm. for the left and right lobes and 19 by 19 cm. for the anterior field.

In addition, quinin hydrobromid, 6 grains daily, was given and extraction of tooth roots and any suspicious teeth advised. Subjective improvement was progressively satisfactory. The attacks of palpitation ceased, nervousness disappeared, and the pulse-rate remained around 80. On December 13, 1924, two and a half months after completion of x-ray therapy, metabolism was +12.5 per cent. More frequent determinations during and after x-ray therapy would have been preferable, but were not made because of the expense attached.

In January, 1925 he expressed himself as feeling as well as he ever had. He has been observed at monthly intervals since, but there has been no return of his hyperthyroid symptoms. The sinus arhythmia is no longer present. Metabolism on August 21, 1925 was +9 per cent. At that time the neck measured 35 cm. in circumference, no enlargement of the thyroid could be demonstrated, there was no excess sweating, and no tremor. No medication has been given since January, 1925. Monthly observations, unless otherwise indicated, will be made. This is a very important point in the management of these patients even though they are apparently cured, as recurrences are not unusual.

Case III.—This patient does not fall exactly into the group illustrated by the 2 preceding cases, but rather into a group characterized by frank enlargement of the thyroid in an elderly individual in which there is some difficulty in deciding whether

the goiter which has been present for years is an incidental or the major condition.

The patient, a woman sixty-six years of age, was admitted to the Out-patient Department of the Chicago Memorial Hospital, service of Dr. L. E. Garrison, December 9, 1924.   Her complaints were numbness of the fingers and a dull aching precordial pain.   The former had been present a few years, the latter off and on for twenty years. She had had measles, mumps, chickenpox, and malaria in childhood.   Surgery had consisted of removal of a lipoma of the shoulder and perineal repair. Goiter had been present for years.   Examination showed a nervous individual who cried easily.   There was a peculiar brown pigmentation of the skin around the eyes.   The neck measured $15\frac{3}{4}$ inches.   The heart was not enlarged; tones regular, the first apical somewhat blurred; impulse diffuse, but not heaving. Blood-pressure was 190/90.   Pulse 100.   Temperature and respiration normal.   The liver extended 6 cm. below the costal margin, but was not tender.   The hands were cold and clammy. Slight edema of the ankles.   The lungs were clear.

While the possibility of hyperthyroidism was recognized, the history and physical findings at the time were not sufficiently clear to warrant a positive diagnosis.   Hospital study was advised, but refused.   She was given potassium iodid, and for a time showed some improvement, although the blood-pressure remained high and the pulse ranged from 96 to 118.

She finally consented to enter the hospital, and was admitted to the Medical Group "B" service March 2, 1925.   At that time she gave an entirely different history.   Her complaints were palpitation, dyspnea on exertion, weakness, hypertension, nervousness, tremor of the hands, choking spells, precordial pain, marked loss in weight, and diarrhea.   These symptoms were verified by her daughter.   It is hard to account for the difference in the two histories, as the same intern took both. Three years previous she was treated for hypertension and nervousness and was considerably improved for two years.   Then dyspnea and precordial pain on exertion began, followed by the loss of weight and other symptoms.

Examination at this time showed slight staring expression of
the eyes, suggestive Von Graefe, Stellwag, and Moebius signs,
marked emotional and vasomotor instability, tremor of the
hands, and excessive sweating. The thyroid felt distinctly
cystic, especially the isthmus. The heart was not enlarged.
A blowing systolic murmur was present over the entire pre-
cordium, heard best at the base, and the second pulmonic sound
was accentuated. Rhythm was regular. Pulse-rate 88, recum-
bent, rose to 110 on slight exertion. The vessels felt thick, but
not unusually so for her age. Blood-pressure was 160/88. The
liver was not enlarged and there was no edema of the extremities.
Respirations were 26, but the patient could lie flat without dis-
comfort. There was no cyanosis. The urine output was moder-
ately diminished, specific gravity 1.020, hyaline and granular
casts and a trace of albumin were present. The metabolism was
+47 per cent., which reading was verified. x-Ray of the chest
showed a substernal thyroid, dilatation of the aorta, but no
cardiac enlargement.

The more pronounced hyperthyroid findings at this time were
not due to difference in care or skill of examination, but, I be-
lieve, to the actual increase in toxicity in the three months which
elapsed from time to admission to the Out-patient Department
and admission to the hospital. The whole picture was not suffi-
ciently clear even then to make one dogmatic about a diagnosis
of hyperthyroidism. Had there been more evidence of cardiac
decompensation the metabolism could have been explained on
that basis. As it was, this factor together with the other findings
made some of us feel that we were dealing with a hyperthyroidism
with coincident cardiovascular-renal changes incident to the age
of the patient. Others felt that the latter condition was primary
and should be the point of attack for therapy. Accordingly, she
was kept in bed for a period and proper eliminative and dietary
measures instituted.

No improvement was noted, and surgical consultation was
had, with the decision that the patient was entitled to surgery.
A left polar ligation was done under local anesthesia by Dr.
Peter S. Clark. She stood this very well, and a week later sub-

total thyroidectomy was done under ether anesthesia. Recovery was uneventful and she left the hospital on the twenty-seventh postoperative day feeling "better than she had for years." The emotional instability, heart murmur, excess sweating, and tremor had disappeared. The resting pulse-rate was 72, increasing to 88 on exertion. Blood-pressure was 120/60. Metabolism +26 per cent. Urine showed a trace of albumin, but no casts. Weight 129 pounds.

She has been observed periodically since. On September 3d the blood-pressure was 140/70, pulse 76, respirations 16, weight 149 pounds. Heart tones were clear. Liver was not enlarged. No edema. Metabolism was −10 per cent. She felt "fine," was not nervous, and able to do full housework.

Here the proof of the diagnosis is found in the subsequent course. Such marked improvement could hardly be expected in a patient having a purely cardiovascular disorder, especially after the insult of a rather prolonged ether anesthesia and surgical procedure.

Discussion.—All 3 of these patients presented symptoms which were primarily cardiac in nature. In the absence of goiter and characteristic eye signs a differential diagnosis is not easy unless one bears in mind certain factors. The appearance of these patients is characteristic. As pointed out by Levine and Sturgis, they are restless, hyperactive, have an alert, animated expression or a constantly startled look. The eyes are apt to be roving and are usually moist. This is in contradistinction to the apathetic expression seen in most patients with other forms of heart disease. Inability to concentrate, subjective nervousness, and emotional instability are common. Symptoms often date from some undue emotional or physical strain (Case II). The paroxysms of palpitation are as apt to come during sleep or while resting as following exertion. Loss of weight and strength in spite of a normal or increased appetite is common. In other forms of heart disease anorexia is the rule. So far as has been reported, all of the cases of this "latent" type of hyperthyroidism have been in middle-aged or elderly individuals.

In attempting to establish a guide to the recognition of the

underlying hyperthyroidism certain characteristic cardiac find-
ings may be mentioned. Attacks of paroxysmal tachycardia,
auricular flutter or fibrillation, or a marked sinus arhythmia in
an adult should make one suspect hyperthyroidism until defi-
nitely ruled out. A pulse-rate showing wide variations, es-
pecially with changes in emotional state and which is not af-
fected by rest or digitalis, is a suggestive finding. The heart
action is best described as tumultuous, with diffuse, heaving
apex impulse, sometimes felt as a distinct thrill or shock, which,
together with the character of the first tone, may lead to an
erroneous diagnosis of mitral stenosis. A systolic blow heard
over the entire precordium, varying in intensity, is common and
may lead to a diagnosis of valvular disease. In hyperthyroidism
this murmur disappears following proper treatment (Cases I
and III). The pulse is likely to be Corrigan in type, but without
other evidence of aortic insufficiency. The vessels of the neck
tend to engorge and pulsate visibly. Such a condition does not
occur in other forms of heart disease except when advanced de-
compensation with failure of the right heart is present. Blood-
pressure is a variable factor. Case II showed a normal or slightly
lowered systolic pressure with a low diastolic pressure. The re-
sultant high pulse pressure together with a rapid pulse is fre-
quently seen in hyperthyroidism and is a suggestive finding.
The systolic pressure may be high, with a normal or subnormal
diastolic pressure. If hypertension is present, both systolic and
diastolic pressures are elevated (Cases I and III), the pulse
pressure being high (Boothby and Plummer, Oxford Medicine,
Vol. III). At the time of recurrence in Case I the second type
was present. Vasomotor instability, excess sweating, and tremor
of the hands are suggestive findings, although these may also be
found in various neurasthenic conditions. Perhaps the most
striking feature of these cases is that the intensity of the cardiac
symptoms is out of all proportion to the signs of actual cardiac
failure.

The basal metabolism test, properly applied and correctly
interpreted, offers the best means of arriving at a correct diag-
nosis in these cases. One hesitates to advocate, as a routine

measure, any more laboratory procedures than are already
deemed necessary to a complete examination of a patient. How-
ever, in dealing with cardiac patients, the test is invaluable
both from the standpoint of ruling out a possible hyperthy-
roidism and as a measure of cardiac decompensation. While
it is true, as shown by Peabody[3] and more recently by Ham-
burger and Lev,[4] that patients with cardiac decompensation
will give a metabolic reading above normal, it is only in the most
advanced cases that the reading approaches the elevation char-
acteristic of hyperthyroidism. A single plus reading does not
make the diagnosis of hyperthyroidism, but if the reading is
persistently high on repeated tests, and especially after a few
days' rest in bed, such a diagnosis is practically certain.

Electrocardiograms are useful, though not essential. Willius[5]
and others have called attention to an abnormally wide deflec-
tion of P and T as characteristic of hyperthyroidism (Fig. 247, B
and Fig. 248). That this is not always present at first (Fig.
247, A), but may develop later (Fig. 247, B), then to recede
(Fig. 247, C) as the thyroid condition is corrected is shown by
Case I. The tracings are also of value in determining the type
of irregularity (Fig. 248, Case II) and in estimating the extent
of cardiac damage.

Most cases of the type here described fall into the group
called by Plummer "adenoma with hyperthyroidism" and by
others "toxic adenoma." A history of goiter in early life which
later receded can sometimes be obtained. In others (Cases I
and II) the patients have never been aware of any thyroid en-
largement, although close inspection shows what appears to be
a slight diffuse enlargement of the gland. The adenomata may
be so small that they cannot be palpated (Case I). In this
case the microscopic sections showed definite small colloid ade-
nomata with some areas of hyperplasia outside of the adenomata.
The colloid was poorly stained. The gland removed at the second
operation was diffusely hyperplastic with small acini containing
little or no colloid. The tissue from Case III did not coincide
with the clinical picture of hyperthyroidism. Sections showed
cystic colloid goiter without hypertrophy or hyperplasia. Atrophy

and degeneration of the parenchyma were present. The colloid stained deeply. This is probably one of the rare instances mentioned by Plummer in which a comparatively mild clinical hyperthyroidism is associated with practically normal microscopic appearance of the gland.

Treatment of this type of hyperthyroidism does not differ from that of the more easily recognized forms. Nearly all being of the adenomatous type, and of some duration when first recognized, operation is the method of choice. If severe cardiac decompensation is present, preliminary rest and digitalization is indicated. Pre- and postoperative digitalization is especially beneficial when auricular fibrillation is present, as it appears to favor a resumption of normal rhythm after operation. Digitalis does not affect the pulse-rate in the cases uncomplicated by cardiac failure (Case I). As has been mentioned, this is of value in the differential diagnosis. The effect of iodin in reducing the pulse-rate and metabolism is striking, and not only permits of earlier surgical interference, but makes the patient a better surgical risk. During the administration of iodin careful record of the pulse-rate and subjective symptoms must be kept and frequent metabolism determinations made in order that operation may be done as soon as the maximum benefit has been obtained. Otherwise the patient may quickly show a recurrence of symptoms. This effect of iodin is another valuable "therapeutic test."

Quinidin is sometimes serviceable in controlling the paroxysms of tachycardia, whether simple or due to fibrillation or flutter.

x-Ray should be tried in those patients who refuse operation, and may be used in the milder cases not showing true cardiac damage. Improvement should be noted after three or four exposures. If such is not the case, or if evidence of cardiac damage becomes more pronounced, operation should not be delayed. Clinical improvement must be checked by frequent metabolism determinations. x-Ray was of no benefit in the recurrent stage of Case I, and some who saw her felt that her condition was made worse by it. It produced at least a temporary cure in Case II. Quinin hydrobromid is a useful adjunct to x-ray therapy.

CONCLUSIONS

1. In certain cases of hyperthyroidism without goiter or exophthalmos the cardiac symptoms and findings may lead one to overlook the underlying condition.

2. The differential diagnosis becomes easier if one will bear in mind certain characteristics of the hyperthyroid heart, and will look carefully for other signs of hyperthyroidism. As a routine examination in the study of heart patients metabolism determinations are of greatest benefit in recognizing a possible hyperthyroidism.

3. Because proper therapy offers complete relief of the cardiac symptoms in nearly every case, the importance of the early recognition of the underlying condition in this type of "heart disease" becomes apparent.

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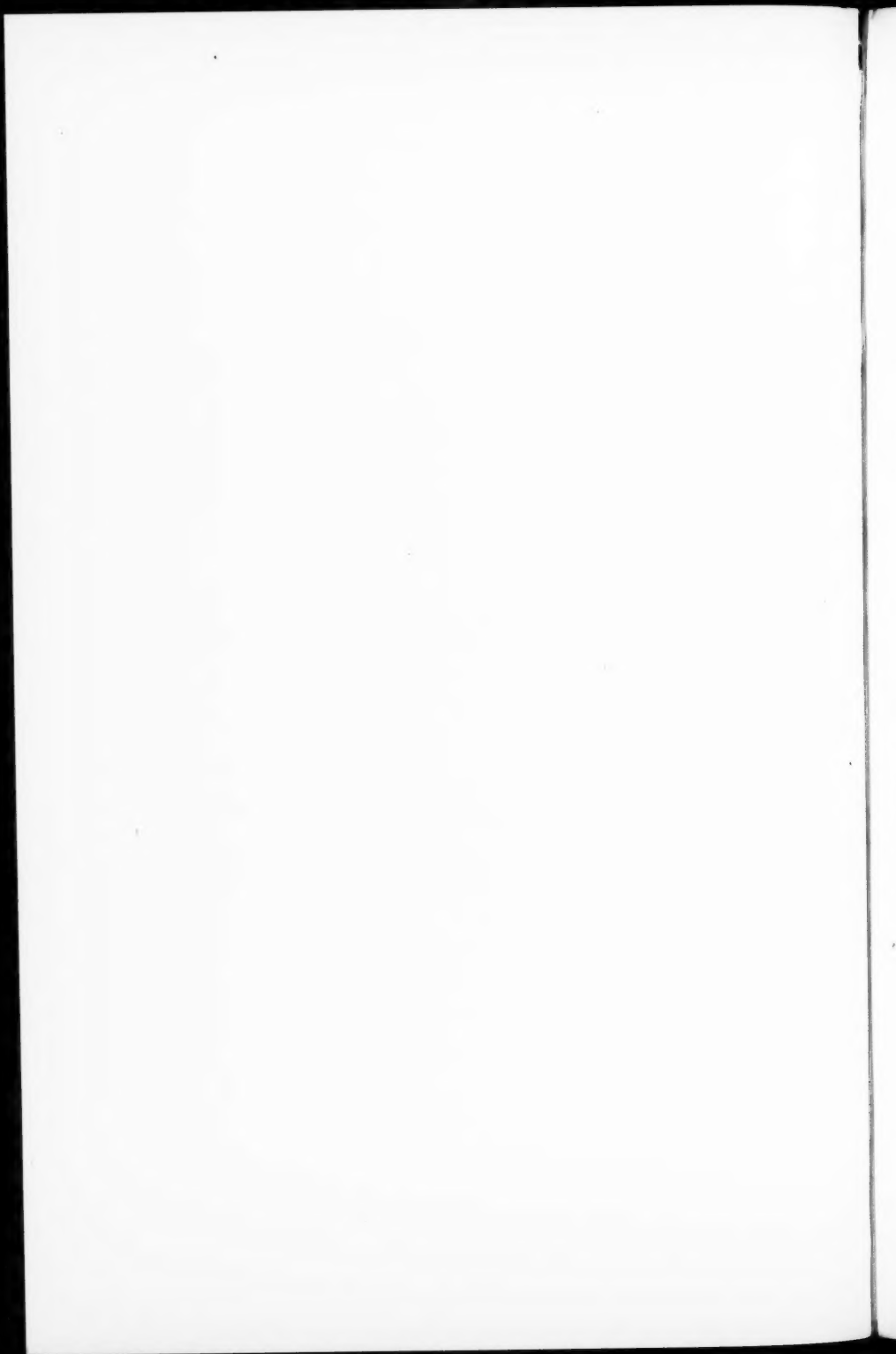
I. A CASE OF PURPURA HÆMORRHAGICA—FULL DISCUSSION OF BLOOD FINDINGS AND CLINICAL FEATURES.

THE first patient to be presented today is a young American girl, twenty-one years of age, who was sent in to us from another hospital with the diagnosis of scurvy. On admission the symptoms complained of were the following:

Present Complaint.—A tired feeling, with great general weakness and rather profuse bleeding from the gums, especially in the region of the right upper premolars. She had noticed that her color was very bad and that there was a general sallow, yellowish tinge to the skin. Two days before her admission she noticed blue spots on both lower extremities, and on the lateral aspects of both shoulders, on the anterior portion of the chest, and quite a large one on the inner side of the lower lip. She complained chiefly of shortness of breath. While the blue spots had only come out a couple of days before her admission, the other symptoms had come on gradually and dated back, she thinks, two or three months.

Onset and Course.—Patient states in the past year she has not had a proper diet, and had had almost no fresh vegetables and comparatively few fruits. These were due to circumstances over which she had no control. Approximately two months ago she commenced to be so tired and worn, especially after coming home from a day's work, that she had to stop work for several days at a time to accumulate sufficient strength to carry on.

Two days before admission she noticed the bleeding from the gums. Before this she had had quite a little pain, which she

attributed to an ordinary toothache. There was some tenderness in that region, but it was not very marked, and a few hours after the pain began a sharp hemorrhage occurred. She tried to arrest the bleeding by hot water applications and later by applying boric acid solution, but the gums continued to bleed until the patient entered the hospital. At present her chief complaints are shortness of breath and the subcutaneous hemorrhages already referred to. These, as you will see from examination of the legs, are very numerous, some hundreds at least, dark purplish in color, irregularly distributed, and not disappearing on pressure. They are obviously hemorrhages into the skin. While the individual spots are small, they take up in the aggregate a considerable portion of the skin of the lower extremities.

On the right side of the neck are numerous subcutaneous hemorrhages in various stages of absorption, bluish and purplish in color, and, on the average, about the size of a quarter.

The patient states that she has not lost any considerable amount of weight; indeed, she is rather inclined to think she has gained a little.

General and Negative.—Cutaneous: Aside from the petechiæ already mentioned and the general yellowish pallor of the skin there are no pathologic changes in the skin. The mucous membrane of the mouth is pale, and a fair sized hemorrhage into the mucous membrane of the lower lip is readily seen. Circulatory: There is no cyanosis and no edema.

Nervous Symptoms.—These are entirely negative and the patient complained of nothing except the tendency to faint.

Gastro-intestinal: Her appetite is poor, but bowels are regular, and she has no evidences of dyspepsia of any type.

Respiratory: Aside from the dyspnea complained of, there are no symptoms pointing to involvement of the respiratory organs.

Past History.—Medical: She has had measles and bronchitis, but no other infections. She has had no surgical or venereal troubles.

Menstrual History.—Shows that she began to menstruate

at fifteen years of age and has always been perfectly regular. Menstruation lasts five days. She has pain during the first day only, and the amount of blood lost is quite moderate and mostly on the first day.

Her habits are quite regular. She sleeps well, and eats regularly in spite of the poor appetite. She has worked steadily and apparently the work is not of a very hard nature.

Family History.—Her father died when she was thirteen. Her mother ran away with another man and left 4 daughters, all well under age, without any supervision, so that our patient has had to assume as best she could the care of the younger children.

Physical Examination.—As we look at her I think you will all agree that she looks sick. Her skin is of a somewhat yellowish hue. The scleræ are of the bluish tinge, commonly known as skimmed milk, and she looks worn and a little haggard.

The examination of the scalp shows nothing abnormal. The eyes react to light and accommodation. The pupils are equal and regular and the muscle functions are good. Ears, nose, and throat show nothing out of the ordinary except for a slightly enlarged pair of tonsils.

Mouth: In view of the history, the examination of the mouth is of considerable importance. As you see, the teeth are in surprisingly good condition, much better than one would expect in the average patient in this stratum of society. They bear unmistakable evidences of fairly good care. There is no sponginess of the gums and no evidences of pyorrhea. On the upper right side you can see a little blood oozing from somewhere, but just where is difficult to determine. As was stated in the history, when she came in, the bleeding was quite profuse, and yet the actual source was not quite apparent except that it was from the gums. When she clamps her teeth together there is no especial tenderness, and pressure on the gums fails to show any sponginess or to cause any exudation of pus or blood except at this one spot.

Neck: There is no adenopathy. The thyroid shows a slight, soft enlargement.

Chest: The chest is well developed. Supra- and infra-clavicular fossæ are normal and both percussion and auscultation of the lungs fail to show anything pathologic. The apex-beat is inside the midclavicular line at about the fifth interspace, two fingers wide, moderately forceful. There is no thrill. Heart borders are normal and auscultation shows no abnormalities.

Abdomen: Well developed, no evidences of free fluid; no tumor masses. No tenderness over the appendix or gall-bladder region. There is slight pressure tenderness over the umbilicus which seems more or less diffuse. This is only to be elicited on deep pressure. The liver and spleen are not palpable.

Genito-urinary: This examination was made yesterday and disclosed nothing out of the ordinary. The inguinal glands are not enlarged. Bones and joints show no limitation of motion and there are no evidences of arthritis or hemorrhages into the joints. There was no tenderness on pressure over the superficial bones.

The neuromuscular apparatus is quite normal. There was some difficulty in eliciting the patellar reflexes on admission, but, as you see, now they can be brought out, with a little patience, by reinforcement.

The blood-pressure is 114/68.

As I mentioned at the beginning, the patient was sent to us from another hospital with the diagnosis of scurvy, as it was known that we had been especially interested in this disease. Let us examine into the patient's condition and see whether this diagnosis is justified by the facts.

In her history she states that she has not had fresh vegetables very often and that she has eaten comparatively little fresh fruit, largely because of poverty. More careful questioning elicited the fact that while they have canned goods a good deal, she has had fresh tomatoes and salads occasionally and canned tomatoes very often. I hardly need to remark that this question was put because of the well-known antiscorbutic value of tomatoes. As you probably know, the antiscorbutic power of this particular vegetable is less affected by heat than most others. She gets milk fairly often.

Now as to the condition of the gums, they are substantially normal except for the fact that there has been a fairly sharp hemorrhage from them, which is still continuing to a slight degree. In particular, there is no sponginess. I am accustomed to compare the gums in a case of scurvy to the red plush that used to be seen so often in our Pullman cars, the color being a purplish-red and the texture distinctly soft and spongy. These gums have absolutely none of those attributes, and on the basis of that alone I should be greatly inclined to question the diagnosis of scurvy. In regard to her diet, this is more difficult to judge. We see quite a good many cases of scurvy each year, mostly in middle-aged men who are out of work and living around at the lowest grades of restaurants or in "flop" houses. Almost all that I have seen have lived for a considerable period of time on bread and coffee or doughnuts and coffee. This has become almost a by-word in the hospital. In previous clinics I have shown you several such cases, and a reference to them will, I am sure, disclose that almost their only diet is that which I have just mentioned. While it has not been very definitely or rather very quantitatively proved, it seems clear that there is some reserve store of vitamins of the antiscorbutic group, so it requires a very considerable, perhaps a nearly complete, deprivation of them to initiate a case of scurvy. Perhaps the young individual has a greater supply of these than the middle aged or old. However that may be, aside from infants and young children, I have not often seen scurvy in young girls. While her diet has been far from a good one, I am a little skeptical as to there having been such a complete absence of the antiscorbutic vitamin from it as to produce a scurvy.

In addition to this, there is another factor lacking which, while not always present, is so generally found as to make us question the diagnosis in its absence. I refer, of course, to the so-called scurvy sclerosis which is nothing more or less than the resultant of hemorrhages into the great muscle masses. I have generally seen them in the thigh and calf muscles, and they are not infrequently so severe as to extend from Poupart's ligament down to the ankle, causing great tenderness and pain on pal-

pation of the muscle masses. As you will see, there is not the slightest sign of this in our patient.

On the basis of the physical examination alone, then, we have really no convincing evidence of the existence of scurvy, and the history, while suggestive, does not compel such a diagnosis. To me the normal condition of the gums is decisive, and on that ground I think we may exclude scurvy. On the other hand, if this is not a case of scurvy, what else would come into consideration? We might first think of some severe infection of a hemorrhagic nature. The patient has had no elevation of temperature at any time since she has been under our observation, and so this, of course, militates very strongly against the diagnosis of the hemorrhagic variety of one of the acute diseases, such as scarlatina, variola, etc. The heart findings are not such as to make us suspect the hemorrhages which are sometimes seen in endocarditis. The other conditions which come into serious consideration are hemophilia, acute leukemia, aplastic anemia, and purpura hæmorrhagica. Now hemophilia, as is very well known, is an exclusively hereditary disease and, according to the best authorities, confined to the male sex. It would require, therefore, the most convincing and thoroughgoing evidence from the examination of the blood and the history to warrant such a diagnosis. Acute leukemia and aplastic anemia will be discussed later.

This leaves us with purpura hæmorrhagica as the thing to be next considered. From the history alone this diagnosis is quite probable, since purpura hæmorrhagica is very common in young girls at about this age and comes on in just about this way. When I say that it is very common in young girls I do not wish to convey the impression that it is a common disease, for it is not. One or two cases a year is about as many as most men see even when they have access to a large clinical material. The gum condition in purpura is quite normal, and there is no sponginess even when the hemorrhages from the gums are quite profuse. In other words, the condition is a hemorrhage from a practically intact gum, and not, as in scurvy, an oozing from a fetid, spongy, swollen tissue with the teeth so loose as to be almost ready to

drop out of themselves. I wish to call particular attention to this because *there is a rather wide-spread but erroneous idea that the bleeding from the gums is the essential factor in scurvy.* This is not at all the case. The sponginess and the stomatitis are the essential factors.

Let us now turn to the laboratory side of this case and see what help we may derive from that side. The blood examination, made shortly after entrance, disclosed 3,550,000 erythrocytes, 75 per cent. hemoglobin, 5400 leukocytes. The differential count showed 74 per cent. polymorphonuclear neutrophils, 18 per cent. small lymphocytes, 6 per cent. large lymphocytes, 2 per cent. transitionals. Blood-cultures were negative. It was noted under the microscopic examination of the blood-smear that a good many vacuolated red cells were present. A further note is made that *the platelets seemed increased.* The stain used was a Wright stain, and I ask your particular attention to the estimate of the blood-platelets that was made as compared with the actual platelet count which we have later. The blood chemistry is substantially normal. The urine showed nothing abnormal.

A very important differential point in the various hemorrhagic diseases is the determination of the bleeding time. Duke's method was used, which is simplicity itself. A small incision is made in the ear and at thirty-second intervals a piece of blotting-paper is touched to the drop of blood which forms, and this operation repeated until bleeding ceases. The normal time is about three minutes. The bleeding time in this case on the first determination was nineteen minutes. It is always wise in making a test of this kind to check it up against a known normal. This was not, however, done the first time. After being in the hospital ten days the patient wished to leave for a few days, and did so, only to return three days later in a worse condition. She came in bleeding again very freely from the gums and from the vagina. Her menstrual period would normally have begun about this time, and she states that she has bled many times as much in the past two days as she ordinarily would have done. This coincides with the estimate of my intern who examined

her on readmission, and I think we must consider the hemorrhage as being definitely pathologic in character.

A few additional cutaneous hemorrhages were evident under the skin, but otherwise her general condition was the same as when she went out a few days ago.

Blood-platelets: The platelet count was made the first time two days ago and was found to be 60,000. In view of the very unsatisfactory state of our knowledge of the platelets and of our methods for counting them, it is well to check up such a count with one or more normal bloods. By the method which we used the normal was found to be about 300,000. Dr. Birch has had considerable experience with platelet counting and has obtained consistently uniform results on the normal cases used as controls. Now this diminution of the platelets is perhaps the most characteristic single feature of the blood in purpura hæmorrhagica. Not only this but the severity of the case or its condition at a given time bears something like a direct proportion to the number of the platelets at that time. Inasmuch as the methods of counting in ordinary use are by no means as accurate as an ordinary erythrocyte count, only large variations in the number of platelets should be used in diagnosis. In a general way we may say that a platelet count of 60,000 or 65,000 represents the number at which we may expect hemorrhagic phenomena, that is, either hemorrhages into the skin and mucous membranes or hemorrhages from the various organs.

A day or two after her readmission blood was found several times in the stools, but as the patient was still menstruating it was not absolutely certain whether the origin of this blood was from the bowel. You will remember that the patient had some tenderness over the umbilical region, and it may be the case that this is to be brought into relationship with the possible blood in the stools. From the diagnostic standpoint this low platelet count is very important in the differentiation from scurvy, since the platelets in scurvy are normal or substantially so in number. The bleeding time has been taken again, and this time found to be forty-five minutes. This is, of course, a very great increase. This greatly increased bleeding time and the low number of

platelets is quite sufficient to exclude scurvy, although we had already decided that scurvy was highly improbable from the physical examination.

I am showing you the blotting-paper charts (Figs. 249, 250) of the bleeding time, and, as you can see, especially when you compare it with a normal chart, the time is greatly prolonged.

Another important test to be made in all hemorrhagic conditions is the determination of the coagulation time. The methods of determining coagulation time are various, so that it is not practical to express the time in minutes unless the method is known, and especially unless one has a normal control. In our patient

Fig. 249.—Bleeding time. Duke's method. Normal control. Total bleeding time, three minutes.

the capillary tube method was used, and the coagulation time was found to be very slightly prolonged. Equally important is the study of the clot. A few cubic centimeters of blood should be drawn into a test-tube, preferably with a syringe, which has been washed out with an ether-petrolatum mixture, the ether being allowed to evaporate, and the syringe thus being coated with a thin layer of the oil. This blood is expelled carefully into a test-tube and allowed to clot. In a normal blood, after the lapse of some time, a firm retractile clot is formed which, by virtue of its firm contraction, squeezes out the serum. This procedure was carried out on our patient, and you see the results in the

tubes before you. The clot is soft, has not retracted, and has not squeezed out the serum. The control tube from a normal individual was done in the same manner, and you will see a striking contrast. The clot is firm, small, withstands considerable agitation, and the serum is clear and can be readily poured off from the clot.

Fig. 250.—Bleeding time. Duke's method. Purpura hæmorrhagica. The drops were taken at one-half-minute intervals, the total bleeding time being forty-five minutes.

Another important test is the prothrombin test of Howell. This is carried out in the following way: Two c.c. of blood are taken, and to this is added 0.25 c.c. of potassium oxalate solution (1 per cent.) and the mixture then placed in the centrifuge tube, and centrifuged until the corpuscles are completely sepa-

rated from the supernatant plasma. A row of small test-tubes is then set up, and each one is charged with 5 drops of this plasma. A calcium chlorid (0.5 per cent.) solution is now added in increasing amounts to these tubes, beginning with 1 drop, and increasing 1 drop in each tube.

The tubes are then inspected at short intervals, and the point at which coagulation first occurs is determined. It is always desirable, as you will see we have done in this case, to set up a row of tubes as a control, using normal blood. In the patient before you the clotting time, or prothrombin time as it is called, was substantially the same in our patient as the normal control.

These tests give us a sufficient basis, I think, for making a definite diagnosis.

Let us begin with acute leukemia. This is sometimes an extremely hard diagnosis, but in our case it is easy. To begin with, acute leukemia sets in with the evidences of an extremely severe disease. The spleen is generally enlarged, and the peripheral glands as well. The blood-count in the acute lymphatic type would show a preponderance of the lymphocytes, whereas in our case we have 70 per cent. of the polymorphonuclears. An acute myeloid leukemia would show a corresponding increase of the bone-marrow cells, which are lacking in our patient's blood.

The differential diagnosis from aplastic anemia is a little more difficult. This disease shows a tendency to hemorrhages into the skin and mucous membranes. The blood-count is very low, in this respect being on a parity with the usual form of pernicious anemia. The color-index is low, whereas in our patient the color-index is approximately 1. The leukocyte count is low in aplastic anemia, and the lymphocytes relatively increased, generally amounting to 80 or 90 per cent. This disease is practically always fatal, and the patient goes down hill rapidly. The platelets are reduced to a very low point in aplastic anemia, as one might infer from the strong hemorrhagic tendency in this disease, so that we do not gain any information of diagnostic value from the platelet count. When we consider, however, the normal color-index, the very moderate reduction only of the erythrocytes, the relatively good condition of our patient, and

particularly the predominance of the polymorphonuclear cells, we can, I think, with perfect certainty eliminate aplastic anemia from consideration.

Now, how do matters stand in regard to hemophilia? Aside from the fact that hemophilia is an exquisitely hereditary disease, it attacks, according to our best authorities, only males. But there are a number of other points in which our patient differs from a patient with hemophilia. The coagulation time of the blood is normal in this patient, whereas in hemophilia it is greatly delayed. Just the opposite is true with the bleeding time. The bleeding time is greatly prolonged in our patient, and is normal in hemophilia. On the other hand, the clot is firm and retractile, although the coagulation time is delayed in hemophilia, whereas in our patient the clot is soft, non-retractile, with a normal coagulation time. From the clinical standpoint the hemophiliac may develop hemorrhages into the joints, but not into the skin. Lastly, the prothrombin time is greatly delayed in hemophilia, whereas in our patient it is approximately normal. From all of these reasons, therefore, we can eliminate hemophilia from diagnostic consideration, and that leaves us purpura hæmorrhagica to be considered.

As a matter of fact, this is a perfectly typical case of this variety of purpura and has all the earmarks of the disease. To summarize the symptoms, we find that it occurs in young girls most frequently, and after a few weeks of prodromes, lassitude, spontaneous hemorrhages in the skin and mucous membrane develop just as in our patient. In addition, bleeding from the uterus, stomach, bowel, and kidneys may occur. An anemia develops rapidly, which is in part consequent upon loss of blood, but, for the most part, not so associated. An important clinical feature is the ease with which subcutaneous hemorrhages occur from slight traumata. The application of a tourniquet will produce hemorrhages into the skin, as occurred in this case while the blood-pressure was being taken. In some cases there is fever. The essential features in the blood itself are, first, a normal coagulation time, or at the most a slight prolongation; second, a very greatly prolonged bleeding time with an absence of the re-

traction of the clot, or at best an imperfect retraction occurring after a very long time; third, a great diminution in the platelet count.

Occasionally, if there has been considerable hemorrhage, a secondary leukocytosis might occur.

Two weeks later: One or two points of additional interest may be noted in our purpura patient. We have put her upon the only form of treatment which promises anything, namely, transfusion of blood. For various reasons we decided to use citrated blood intramuscularly, using 30 c.c. at each injection. She has had three or four injections, and I think you will be interested to know the effect of these. Before the last injection her platelet count was 175,000. In a few hours it had risen to 350,000. It remains, of course, to be seen whether such a rise will last. I have very grave doubts about it, as the platelets are very evanescent structures. Another interesting point is that we have had several of the sisters come in, and determined their platelet count, to see if there were any familial tendencies. In one of them, a big strong girl, the count was 370,000; in the second, rather undernourished and frail, it was 260,000. As a matter of interest rather than of diagnostic importance we have determined the fragility of the red cells and found that hemolysis began at 0.34 and was complete at 0.3 per cent., that is, at approximately the normal point, certainly not any increased fragility.

In regard to the prognosis no very definite statement can be made. The condition tends to recur. Some cases do and some do not get well. We have made every effort to improve her hygienic conditions, and are having her come in at weekly intervals for intramuscular injections of blood.

In view of the tendency to get alternately better and worse, we must carefully guard against drawing any conclusions from the results of therapy in an individual case.
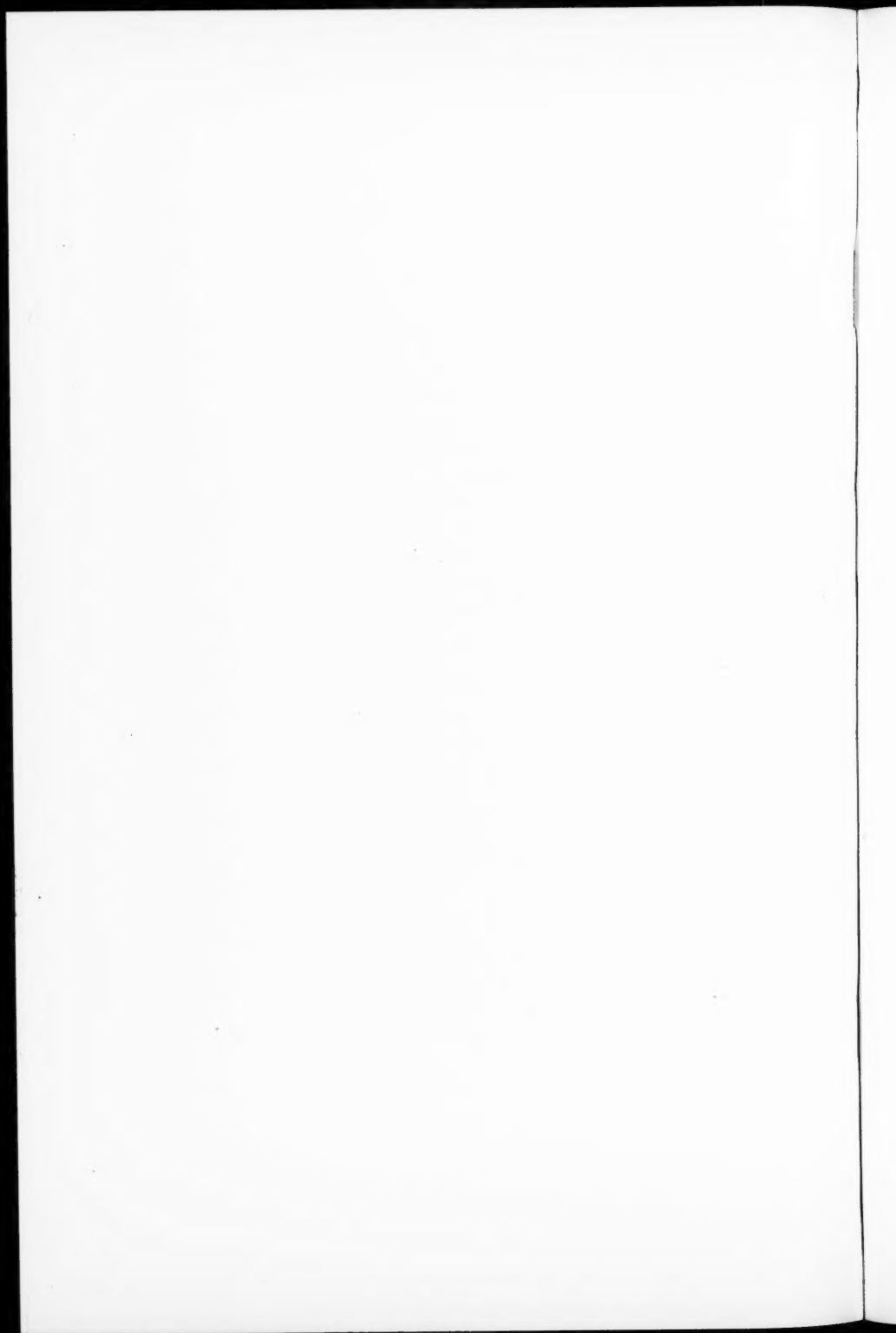
II. A CASE OF LEAD ENCEPHALOPATHY MIMICKING AN ACUTE ABDOMINAL CONDITION

THE second patient I have to present today is a young colored man thirty-two years of age, who came into the hospital on October 1st. His complaints were pain in the abdomen, nausea, and vomiting, with great tenderness over the abdomen, especially in the lower right quadrant.

Onset and Course.—A few days previously he states that he was at work in a factory when he was suddenly seized with a violent cramp-like pain in the lower right abdominal quadrant. The pain was so severe that he was forced to stop work. He went home immediately and went to bed, where he has remained since. He procured some medicine from the drug store on his way home, and though he took a large bottle of it, it gave him no relief. The pain has been a little less severe during the past two days. Nausea and vomiting began yesterday and persisted ever since. He has been constipated for the past three days, but before that his bowels moved regularly, at least once a day.

Tenderness over the abdomen has been present since the onset and has been growing increasingly acute. Today he seems a trifle better.

A few hours before admission he said the pain spread to the rest of his body. He had eaten nothing for three days previous to admission.

General and Negative.—Aside from the symptoms already enumerated, there has been nothing especially noteworthy in the symptomatology. In particular, there were no symptoms on the part of the lung, no rapid respirations or pain in the chest. Symptoms on the part of the nervous system are entirely negative. Genito-urinary symptoms are absent and he denies venereal infection.

I think, on the whole, that the really important point in connection with this case will be better brought out if, instead of giving you the complete physical examination as we now have

it, I will relate to you exactly how and under what conditions I saw the patient. He came to the ward very late in the afternoon after I had finished my rounds and had on my hat and coat preparatory to leaving the hospital. I was asked to come back and take a look at him, as the diagnosis had been made of acute appendicitis and the question of operation was therefore to be considered. Being under high pressure, I made no examination other than that of the abdomen, and found the following conditions: There was no especial distention, but considerable rigidity over a large part of the abdomen, which was, however, very much greater over the region of the appendix. Deep pressure at this point elicited a great deal of pain and there was some pain over the rest of the abdomen. His temperature was taken while I was there and found to be only 98° F. with a pulse-rate of 70 and respirations of 18. He vomited a large amount of watery material with a great deal of bile while I was examining him. My intern informed me that the examination of the lungs had shown nothing, and my impression of the case was that something acute was developing in the abdomen. Naturally enough, the idea of acute appendicitis was uppermost in our minds, especially so since at the time I saw him we had not obtained anything like an accurate history and the patient was in such great pain that both history and examination were unsatisfactory. The patient is at best of a very low grade of intelligence, so it is quite probable that even now the history is not entirely accurate, although we have supplemented it from statements made by his brother. The one thing which was striking was the complete absence of fever, and this was about all which stood in the way of a positive diagnosis of appendicitis. I did not have time to wait for a leukocyte count, and so I left with the direction to ask one of my colleagues to see him if the gravity of the case seemed later to warrant it, and to ask the surgical resident to call up his chief if the case grew much worse.

The further progress of the case is of great interest. When I came over about noon of the following day the patient had taken on an entirely different aspect. The temperature was subnormal, pulse-rate varying from 60 to 74, vomiting had con-

tinued very profusely and occasionally streaked with blood. A blood-count had been made and the number of leukocytes found to be a little over 8000, with a polymorphonuclear count of 71 per cent. The erythrocytes showed a distinct anemia, there being but 3,300,000, with a hemoglobin of 70. The urinalysis was quite negative except that the urine was concentrated, as might have been expected from the amount of vomiting which had occurred. In the meantime a complete physical examination had been made and the points in the history elicited which I gave to you a short time ago. Overnight the patient was complaining bitterly of pain all over the body. The points which interest us especially are that the tenderness had now shifted, so that instead of being in the right lower quadrant it was exceedingly intense over the umbilicus. The patient was still vomiting, the vomitus being bile-stained fluid, and in small amounts. The physical examination now showed the following:

The pupillary reflexes were present, but the reaction to light on the left side was more sluggish than one would normally expect in a young man. The patellar reflexes were absent. With this exception the nervous system showed practically no changes. Needless to say, we immediately thought of a cerebral condition and a lumbar puncture was made. It showed 70 cells per cubic millimeter with, of course, a positive globulin. The pressure was somewhat increased, 88 drops per minute being the rate of flow. The cell count showed 85 per cent. lymphocytes, 15 per cent. polymorphonuclears

Had we stopped here we should unquestionably have made the diagnosis of cerebrospinal lues, and all the more so when the Wassermann was returned 4 + positive. In the meantime, however, another point in the physical examination gave us the real clew to the case, namely, that in the examination of the mouth a very well-defined lead line was found. I say lead line because the line is typically punctate, deep blue, and *in* the gums, not *on the surface*, and these are the typical findings in lead intoxication. A number of years ago in another clinic[1] on the same subject I

[1] A good colored illustration of this line is to be found in this clinic, Medical Clinics of Chicago, Vol. I, No. 3, page 418 (1915).

laid stress upon the punctate character of the lead line, and especially upon the necessity of looking for it carefully with a hand lens, since occasionally only one or two small dots, with difficulty visible to the naked eye, are to be seen. In the meantime the patient had become deeply stuporous, rousing himself only a little and being unable to answer questions very intelligently. His stupor deepened greatly, although at no time could it be properly called profound coma. The patient's brother had in the meantime arrived, and from him we found that the factory he worked in was one in which his duties consisted in handling lead pretty constantly. He had been working in this factory for the past four weeks, as had also his brother. He stated that there had been many cases of lead-poisoning among the employees in this factory, but that his brother was taken sick so violently and so quickly that nobody thought of its being lead-poisoning.

The subsequent history of the patient is exceedingly interesting. He remained in a stuporous condition for several days. The bowels were obstinately constipated during this time. Then he gradually emerged from his stupor and regained his normal mental condition which, as I already indicated, is not much above that of a moron. In the meantime, as you may well imagine, we were thinking quite hard as to the diagnosis. With a normal white count and the shifting tenderness in the abdomen a subnormal temperature and slow pulse, the diagnosis of an acute abdomen could, of course, not be maintained. The next question arose, Did we have to do with a beginning coma of a cerebrospinal lues or with the cerebral manifestations of a lead-poisoning, so-called lead encephalopathy? The very rapid onset of the case, if the history given us by the brother and the patient himself was correct, caused us to lean to the diagnosis of lead intoxication as the cause of his immediate symptoms. We therefore emptied his bowels very thoroughly by means of repeated enemata and purging with salines. I wish to call your very especial attention to the necessity of this preliminary purging before administering iodids. While there is a difference of opinion on the subject, I personally am very sure that on several occasions I have seen patients made materially worse by immediate ad-

ministration of large doses of iodids before getting rid of whatever lead may be contained in the bowel. After two days of thorough purging we started the patient on small doses of sodium iodid, and in another forty-eight hours he was quite rational again and his symptoms had almost entirely subsided. No antiluetic treatment was given at this time. Examination of the urine showed the presence of minute quantities of lead. While it may be thought that the examination of the urine is of great significance, men engaged in industrial medicine, who have had much greater opportunities of seeing lead cases than we in general hospitals do, find that it is by no means always to be found and is, therefore, unreliable as a diagnostic criterion. I think in view of the fact that the patient emerged very promptly from his mental condition after treatment favoring the elimination of lead from the system, and inasmuch as in the weeks following no subsequent symptoms of cerebrospinal lues had developed, that we are justified in assuming that all of his symptoms were due to the lead intoxication.

I need hardly say to you that the number of lead cases in a great city like Chicago is very considerable. The physicians at the great institutions using lead are thoroughly familiar with these conditions, although even then the sudden onset with a very suggestive picture of an acute abdomen is uncommon. I have only seen 1 or 2 cases of this kind, though many have been described, and operations for acute appendicitis have more than once been carried out on patients of this sort. Almost the only thing which kept us from falling into a similar error was the absence of fever, the normal pulse-rate, and the next morning the low white count. A few words might be said on the subject of lead-poisoning in general, although it is much too large a topic to discuss here. The lead line is absolutely pathognomonic when found, but it must be searched for carefully. I always use a hand lens and look particularly at the gum *on the inside of the teeth*. In this case practically all of the line was in that locality. I have been greatly interested for a long time endeavoring to find just what the histologic picture is in such cases. It was known many years ago, and taught especially by Kolisko

in Vienna, that lead has a predilection for the middle coats of the smaller arteries. The peculiar punctate character of the line which is in a well-developed case very striking leads one to ask whether or not in the very small vessels the lead might be deposited in the loops in the gums. As you will notice, several teeth have been recently extracted because they were in a horrible condition. We had this done, and then my dental colleagues were good enough to snip off some small pieces of the alveolar margin for the purpose of a careful histologic study of the location of the lead. Professor Jaffe has been kind enough to undertake this for me, but the study has not yet been completed.

A very important point in all lead intoxication is that the great majority of the cases come from lead in a dust form rather than in a liquid or moist form. In other words, a painter is not nearly so likely to acquire lead intoxication from the moist paper as he is from sandpapering or scraping off old lead surfaces. A large number of cases are now found in workmen who sandpaper surfaces covered with lead paint, such as automobile bodies, especially where this is done by machinery, using large belts covered with sandpaper to do the actual work. The workmen sit in a cloud of finely divided dry lead paint. There is one additional point of interest in this patient. You will have noticed that he had a scar on the abdomen which came from an old bullet wound. A fluoroscopic and film examination show the bullet still to be present at the level of the top of the sacrum, and it might be a perfectly pertinent query to ask if lead-poisoning could come from a bullet retained in the system. Two or three such cases have been reported, but in view of the history in this case I think the presence of the bullet is of no significance and has nothing to do with the lead-poisoning.

In the way of treatment we have used sodium thiosulphate quite a good deal in the last few years, given in doses of 5 or 6 grains intravenously. I have not been able to see any better results than with iodid. I know of no particular reason why they might not be used alternately or even simultaneously, since the one is given by mouth and the other by intravenous injection.

To sum up, the instructive part of this case is to illustrate a point which is not commonly appreciated, namely, that while the ordinary case of lead colic does not in the least resemble an acute inflammatory intra-abdominal condition, yet occasionally, as in this patient, we may find an acute appendicitis mimicked pretty closely. If fever had been present, as sometimes happens, the diagnosis would have been very difficult. The existence of an old cerebrospinal lues further complicated the matter in this particular instance.
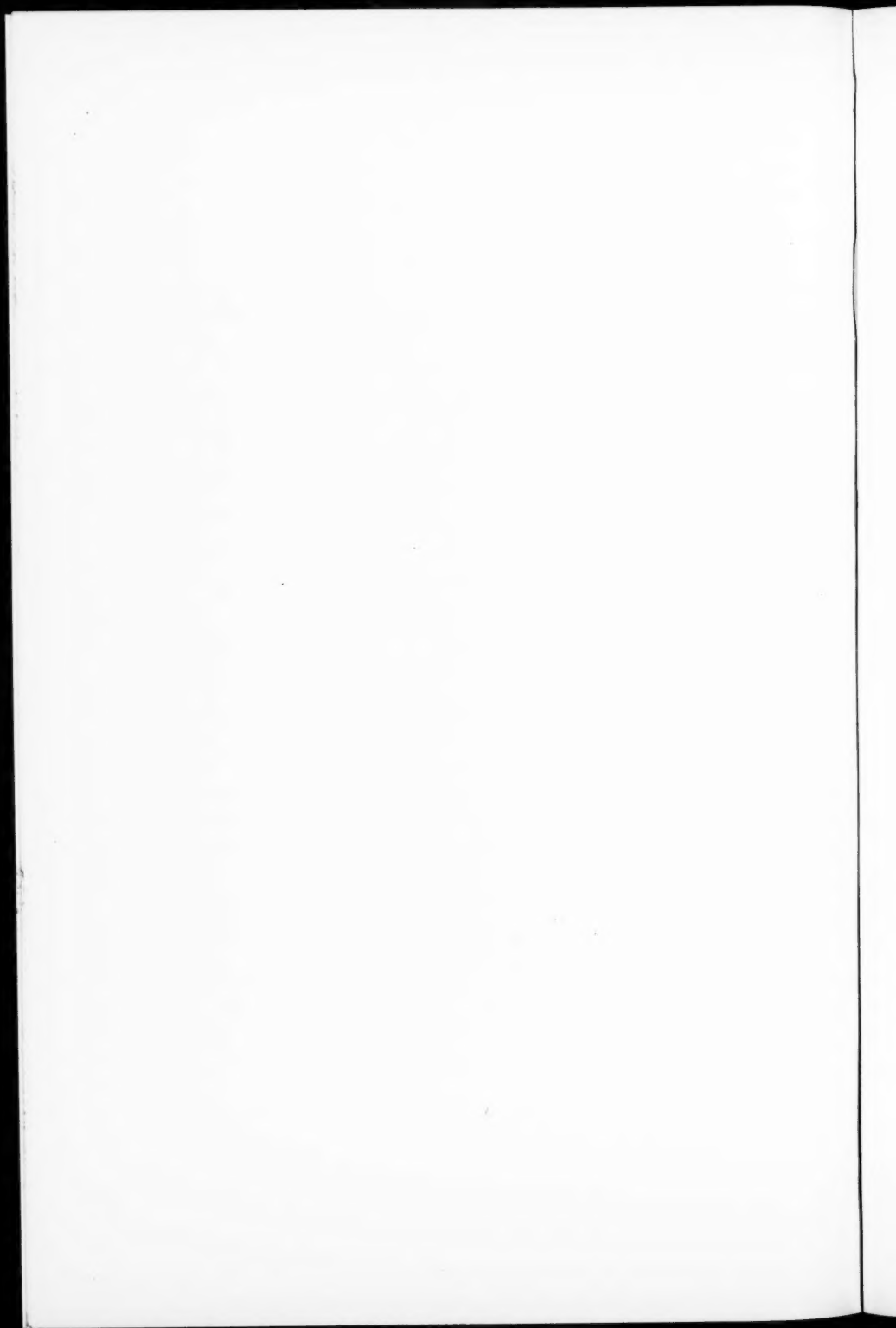
CLINIC OF DR. ROBERT SONNENSCHEIN

A DEMONSTRATION OF EAR, NOSE, AND THROAT CASES

TODAY we will be able to show you a number of cases of ear, nose, and throat conditions, each of which will probably carry a little lesson of its own. No matter how common a condition may be, each individual instance shows a slight deviation from the next one. It is, therefore, best to try to learn some practical points from each case which comes before us in the clinic.

Case I.—*Hemoptysis from Pharynx and Tongue.*—This patient, Mr. J. W. G., age seventy-two years, was in to see us, as you recall, about a week ago. He complained at that time that he had been expectorating blood for many years. There was no history of trouble with his chest nor had he been ill for many years. His occupation is that of a mechanic, and even at this advanced age he is still quite active. You see he is a well-developed man who does not show his age by several years.

Examination of his nose showed simply a deviation of the septum to the right. The pharynx showed submerged tonsils with some secretion in the crypts. There were also dilated veins forming quite a plexus in the mucosa covering the upper pole of the left tonsil. Inspection of the base of the tongue revealed a plexus of dilated veins on the left side of the lingual base. You will recall when we first saw him we were not quite sure whether he had the hemoptysis merely from dilated veins which we have just described. However, at that time cauterization with the red-hot platinum point was carried out both on the left side of the base of the tongue and at the upper pole of the left tonsil.

The patient is a heavy smoker, and in order to avoid irritation of the throat he was advised to reduce his smoking as much as possible. You see him today, and on inspection of the throat you will find the white eschar in the places where the cautery had been applied. He states that thus far there has been no further expectoration of blood since the cauterization.

The causes of hemoptysis or coughing up or expectorating of blood are many. In a large percentage of cases a lesion of an associated tuberculosis of the larynx or lungs is the etiologic factor. It is necessary, therefore, before making a definite diagnosis as to the source of the bleeding to make a very careful inspection of the nose, nasopharynx, pharynx and tongue, larynx, trachea, and lungs. In the nose the bleeding is most often due to some lesion in the anterior portion of the cartilaginous septum either from traumatism, such as picking of the nose with the finger, the insertion of foreign bodies, especially by children or the insane, violent blowing of the nose, operative procedures, or the use of caustic medicaments. Furthermore, systemic conditions, such as high blood-pressure, arteriosclerosis, profound anemia, sudden change to high altitude, or vicarious menstruation may produce bleeding. In many of these cases the blood, instead of running out of the anterior nares, flows along the floor of the nose into the throat and is expectorated; the source is then often regarded as arising either in the throat or trachea, since in many instances no pain or discomfort attends the nasal hemorrhage. A lesion in the posterior portion of the nose or in the nasopharynx may give the same result. We often see dilated veins on the posterior wall of the pharynx, in the tonsillar region, and especially on the base of the tongue over that portion known as the lingual tonsil. It is often very easy to have a rupture of one of these veins, with consequent bleeding and hemoptysis. In the mouth itself it not infrequently happens that the bleeding is from the gums, either due to retraction, to too vigorous brushing, with injury of the mucosa, or to the habit of sucking which some people indulge in, thus producing extravasation of blood from that portion of the gums close to the teeth. If no source of bleeding can be found in the upper respiratory tract or mouth,

it is then most important to make a careful examination of the chest—including x-ray and fluoroscopy.

Where the lesion or source of bleeding is in the nose, nasopharynx, mouth, or throat, local measures may often be used, such as chemical caustics, chemical instillations, like the silver nitrate bead, trichloracetic acid, or chromic acid bead. It must always be remembered, however, that if chromic acid is used a concentrated solution of sodium bicarbonate must be applied at once in order to neutralize the excessive acid and prevent deep ulceration of the adjacent tissues. In many cases, however, the use of the galvanic cautery at dull-red heat, as in our case, gives the best result. Care should be taken to touch with the platinum electrode only the actual bleeding points, and to avoid destroying other portions of the mucosa.

If systemic conditions, such as anemia, high blood-pressure, etc., are present, proper measures for their relief should be instituted by the internist.

Case II.—*Lues Plus Chronic Sinusitis.*—We will now demonstrate to you 2 cases in which the question of luetic infection of the nasal tissues is of great importance. As we have stated on previous occasions in this clinic, we often prefer to use the word "lues" or luetic condition in place of syphilis, since the latter term is so well known to the laity and carries such a stigma with it.

Mr. H. L., age thirty-two, a clothing merchant, first appeared in this clinic some months ago. He stated at that time that four months previously he had developed an eye condition, apparently an iritis, which was thought by the ophthalmologist to be due to some nasal condition. x-Ray films of the sinuses showed some involvement of both ethmoidal labyrinths and also cloudiness of the right maxillary sinus. The ophthalmologist was of the opinion that a nasal operation was indicated, as it would probably relieve the iritis. The patient had a history of only very rare attacks of sore throat. He, however, had frequent so-called "head colds" and some nasal obstruction. For three weeks before his first visit to the clinic he had a ringing tinnitus in his right ear.

Our records show that at that time the case presented a marked deviation of the nasal septum to the left, with a crest on the right side. The middle turbinates were quite boggy and thickened. The tonsils were flat and submerged, and contained a small amount of secretion. Transillumination in the dark room showed that the frontal sinuses were negative, but the right antrum of Highmore was cloudy.

In view of his iritis we were somewhat suspicious of a possible lues, and on that basis had a blood Wassermann made, which was negative. Relying upon this finding we proceeded to operate on his nose. A submucous septal resection was performed, the anterior portion of the middle turbinates snared, and the anterior ethmoidal cells opened. The patient, as you will recall, was greatly relieved so far as his nasal obstruction was concerned, but the eye condition did not improve. He was then given a provocative intravenous injection of a small amount of neosalvarsan, and then blood Wassermann tests were made in two laboratories, both of which reported strongly positive Wassermanns, one 3+ and the other 4+. Intravenous injections of neosalvarsan at weekly intervals and in increasing dosage were now employed. At the same time he also received injections of mercury salicylate into the buttocks. Under this combined treatment the iritis promptly and steadily improved; the eye, as reported by the ophthalmologist, is almost normal at the present time.

A very important lesson is to be drawn from this case. It is well known that operations, particularly upon bony or cartilaginous structures of the nose, are likely to be followed by very poor results if an active lues is present. In many cases, if a septal resection is performed, there is marked destruction later on of the nasal bones or so-called "bridge," which supports the upper or bony portion of the nose. This often gives a sinking in of the dorsum, producing the "saddle nose," an appearance similar to that seen in hereditary syphilis. Therefore, in all cases where an operation is contemplated either on the nose or throat and where there is the least suspicion of luetic infection, even though denied by the patient, we should have one or more

Wassermann tests made. Where it proves to be positive, no operative procedure is to be undertaken until intensive treatment has been given, and the serologic test is repeatedly found negative. In this case, fortunately for us, nothing of an unfavorable nature happened, even though operation was performed while the patient had a latent lues. However, we had not neglected having the Wassermann test made. On the basis of the negative report we had every reason to assume that nothing serious would result, and therefore proceeded to operate. It is well known that a great many cases of lues will show a negative blood Wassermann. If, however, a provocative injection of salvarsan or neosalvarsan is made, then the reaction will usually be positive. Naturally, no one is likely to use a provocative dose unless some very strong suspicion of the presence of lues exists despite the negative test. In our case if destruction of the bone had occurred it would have been most unfortunate, but at least our conscience would have been clear in that we had not overlooked the possibility of lues.

Case III.—*Nasal Syphilis.*—Mrs. D. L., age twenty-nine years, is an old patient of ours. When she first came to the clinic some months ago she complained of having had a severe "head cold" for six weeks, with considerable nasal discharge and difficulty in breathing. The peculiarity of the history was that the symptoms both of discharge and obstruction were mainly present at night time, and accompanied by pain, but only on the left side of the nose. Usually these so-called head colds present bilateral symptoms.

Examination of the nose at that time showed a large left middle turbinate with erosions and a white exudate therein. The left side of the septum was greatly thickened and likewise showed an ulcerated area in its middle third. There was a firm wide adhesion between the septum and the left inferior turbinate. The left naris was almost completely obstructed, so that even with the use of cocain and adrenalin it was impossible to get a view of the posterior half of the nose by way of the anterior rhinoscopy. The pharynx showed submerged tonsils, but no

pus in the crypts. Transillumination of the sinuses was nega-
tive except for some haziness of the left antrum. The appearance
of the nose was rather puzzling at first. In view of the marked
thickening of the septum a blood Wassermann was made and
pronounced negative. Smears were made and examined in an
effort to see if an infection of the Plant-Vincent type were pres-
ent, but none was found.

In view of the fact that gumma of the nose most often in-
volves the septum and that we were at a loss despite our ex-
aminations to come to a definite diagnosis, a provocative in-
travenous dose of neosalvarsan was given. The next blood
Wassermann test was 4+ positive. The patient objected at
first to the use of salvarsan intravenously, since she had very
small veins, and it was with a great difficulty that the injections
could be made. Therefore a few hypodermic injections of
sodium cacodylate were given, but without producing any
favorable effects. The neosalvarsan injections were then re-
sumed, with increasing amounts, once a week for nine weeks.
At the same time she received 10 grains of potassium iodid three
times a day and mercurial inunctions. This combined treat-
ment has had, as you can see, a most gratifying effect. The
swelling and ulceration of the left middle turbinate and left
septum have practically disappeared. All that remains is
the previously mentioned band of adhesions between the sep-
tum and the left inferior turbinate, which is probably an
incidental process having nothing to do with the lesion under
treatment.

Here we see that the same principles regarding the presence
of luetic infection apply as in the previous case. This patient
had no symptoms or even a suspicion of any infection, and her
husband likewise denied having had any specific trouble. When
the possibility of a luetic lesion dawned upon the patient she
became almost suicidal in her attitude, since she considered it
such a terrible disgrace. As a matter of fact, it was probably
quite an accidental infection, and after this was explained to her
she became more reconciled to the lesion and submitted to the
treatment cheerfully and faithfully.

Case IV.—*Nasal Carcinoma.*—Nasal tumors, especially those involving the interior of the nose, are not very common. There are both benign and malignant growths, the benign being the more common. Of the malignant growths, sarcoma is by far more often seen than carcinoma. For some reason or other the malignant growths which arise definitely within the nasal cavity itself usually do not seem to have so rapid a growth, such early metastases, or cause such early cachexia and death as do malignant growths in many other parts of the body. Where the neoplasms affect the exterior of the nose this statement does not usually apply.

This patient, Mr. M. J. I., age sixty-three years, a teacher, presented himself at the clinic a few weeks ago with a history of having had epistaxis for the previous three months, particularly from the right naris. For a few weeks prior to coming here there had been some swelling of the right side of the nose. Eight weeks previous to his first appearance he had a facial erysipelas. There has been no loss in weight, nor has he had any general symptoms which would indicate the presence of a malignant neoplasm.

Examination of the nose showed a deviation of the septum to the left with an erosion on the right side, and a rather large irregular polypoidal mass in the right naris which bled very freely with the slightest manipulation, even on touching it with a cotton-covered applicator. The pharynx showed small flat tonsils. Transillumination of the antra disclosed some haziness of both sides. The x-ray film showed a definite mass in the right naris, both antra somewhat cloudy, but there was apparently no connection between the nasal mass and the right maxillary sinus.

Under cocain anesthesia a few weeks ago the large polypoidal mass was snared from the right naris. At the same time the ethmoid cells were opened. Because of the erosion on the right side of the nasal septum trichloracetic acid was applied to this area. A rubber finger-cot filled with cotton and covered with petrolatum was inserted in the right naris and allowed to remain for twenty-four hours. After its removal there was very little of the much feared and anticipated bleeding.

Histologic examination showed a structure strongly sugges-
tive of carcinoma, with the epithelial cells arranged in alveoli.
Fifty milligrams of radium were placed into the right naris for two
hours.   This patient will require further treatment by means of
radiation, but at present there is quite a free space where formerly
there was a complete obstruction.   The tissues in the region of
the middle meatus bleed very easily when touched with a probe
or a cotton-covered applicator.   It is impossible to state in this
case whether the use of radium will effect a permanent cure.   It
is well known that the mesoblastic tissues are much more amen-
able to radiation, both of x-ray or radium, than are the epiblastic
structures.   It is for this reason that sarcomata, especially the
small round-cell variety, yield so well, as a rule, to radiation,
whereas carcinoma, particularly epithelioma, is often very re-
sistant.   Of the carcinomata those with columnar cells yield
better than does the squamous-cell type.

Case V.—*Cavernous Hemangioma of the Nasal Septum.*—
The next patient whom we will show you presents, fortunately,
a benign nasal growth.   Mr. S. R., age thirty-eight years, married,
a manufacturer, came to the clinic two weeks ago with the his-
tory of having had epistaxis and obstruction to breathing on the
left side of his nose.   This condition had been present for three
or four weeks.   There had been some obstruction to breathing
for about fifteen years, but not to the degree that had been pres-
ent for the four or five weeks previous to admission to the clinic.
There was no history of sore throat.   Examination of the nose
at that time revealed a large polypoidal mass attached to the
left side of the anterior portion of the nasal septum.   This
growth was smooth, red, or purplish in color, and bled very
freely on the slightest contact, even that of a cotton-covered ap-
plicator.   No other abnormalities were present in either nares.
The obstruction to breathing on the left side was entirely due
to the growth on the septum.   Examination of the mouth and
pharynx was negative.   The tonsils were large and partly sub-
merged, but contained no pus.
Under cocain anesthesia this growth was very easily snared

and removed in one piece. Its base was much smaller than the inspection of the tumor at first would have led one to assume. The base bled very freely, but, after pressure with cotton and the application of pure silver nitrate in the form of a bead, the bleeding practically ceased. A rubber finger-cot filled with cotton and covered with petrolatum was inserted in the left naris. On its removal twenty-four hours later there was no bleeding and the patient was allowed to leave the clinic.

Inspection of the mass showed a round body measuring 1 by 1 by 0.5 cm., with completely smooth surface except on its inner concavity, where it was hemorrhagic. On section the interior was quite dense and cellular. Microscopic examination showed that one end of the section, the subepithelial stroma, was pale and edematous; at the other end, which was apparently the attachment of the tumor, there was an area of nodular thickening in which the tissue beneath the epithelium contains many thin-walled blood-vessels lined by more prominent endothelium. The pathologic diagnosis was that of cavernous hemangioma of the nasal septum.

Benign tumors of the septum are rather uncommon, but when present may be fibromas, epitheliomas, angiomas, or chondromas. In the case of the more vascular growths treatment with radium is sometimes very efficacious. On our patient, however, we have not thus far deemed it necessary to use it, because by means of the snare the whole growth was removed. Since this is a benign tumor there is practically no probability of its recurrence. In the case of nasal polpys, which are also growths of the definitely benign type (probably myxomata), we know that recurrence is almost always the rule. In these cases the use of radium has proved very beneficial in the hands of many operators.

The after-treatment in our case consisted merely in having the patient use petrolatum twice a day in order to avoid drying of the secretions, and also to prevent the marked crusts which sometimes occur after operation. Healing has taken place beautifully. The mucosa has grown over the former site of the attachment of the tumor and the patient is probably completely cured.

The last group of cases which we will demonstrate to you are those of mastoiditis with one or another complication.

Case VI.—*Acute Mastoiditis.*—This little girl, F. L., age seven years, presented herself at the clinic about six weeks ago. The history which was elicited at that time was that there has been pain in the right ear following an attack of flu. Tonsillectomy had been performed at the age of two years, but the patient had nevertheless suffered with sore throat one week before admission to the clinic.

Examination of the nose was negative. The pharynx showed flat tonsils, submerged, especially on the right side. In the neck was a number of enlarged lymphatic glands. Examination of the ears showed the left one negative, but injection and bulging of the right drum membrane. Under nitrous oxid gas anesthesia paracentesis of the right drum membrane was performed. The peculiar thing in the symptoms of the next few days was that although there was free drainage from the right ear the pain continued almost unabated. It was necessary at times to give the patient $2\frac{1}{2}$ grains of pyramidon, and that gave only temporary relief. There was no swelling of the mastoid process and there was very little fever; in fact, most of the time the patient was afebrile. Nevertheless the pains in the mastoid region and in the right ear continued with very few remissions. The x-ray films showed the absence of, or at least very marked destruction of, the cells on the right side. In view of the fact that the patient did not improve, that the discharge continued, and that the pain did not disappear, the right mastoid process was opened under ether anesthesia two weeks after the child came to the clinic, and three weeks after the first pains had appeared in the ear. On removing the mastoid cortex it was found that there was very poor pneumatization. There was some pus under pressure in the mastoid antrum. The sinus was exposed and looked gray instead of the normal blue. The child, as you can see, has made a perfect and uneventful recovery.

The interesting and somewhat unusual points in this history are, first of all, the long continuation of pain in the ear despite

paracentesis and free drainage. It is well known that very often some pain continues for twenty-four to thirty-six hours after paracentesis, even though sufficient drainage has been provided, and also that in some cases where there is periosteitis of the bony portion of the external auditory canal or meatus, pain may persist for some days after opening the middle ear. In our case the pains continued for weeks despite the drainage. The cases which are poorly pneumatized, namely, those in which the cells in the mastoid process are not well developed, are much more likely to run an atypical course than are those cases in which complete pneumatization takes place. In the mastoid process during the first year, or at least the first few months of life, there is only one large cell, known as the antrum, and from this point downward and backward pneumatization takes place, so that at the age of seven this formation of air spaces or cells is fairly complete. However, the x-ray picture had already shown that the cells in our case were destroyed or poorly developed, and this was verified at operation. Another very important point was that even though this patient only occasionally had slight fever, still the lateral sinus showed changes in its wall (namely, phlebitis), as evidenced by its gray appearance. Had this phlebitis progressed further there is a possibility that a sinus thrombosis with its consequent pyemia might have resulted. In most cases where there is either a phlebitis of the lateral sinus or a perisinous abscess the temperature is often of a distinct pyemic type just the same as though a definite thrombosis with embolism were present; in other words, the temperature may often rise to 104° or 105° F., either following a chill or without it, and then drop within a few hours to normal or subnormal, to be followed by another chill or rise in twelve, twenty-four, or thirty-six hours, and the cycles repeated until surgical intervention is instituted. In our case, however, as previously noted, there was no temperature elevation at all suggestive of any infection of or in the vicinity of the lateral sinus.

Case VII.—*Double Acute Mastoiditis With Septic Temperature and Followed by Measles.*—Little baby, A. A., age eleven

months, presents a most interesting clinical history. When we first saw him, two months ago, his mother told us that two weeks previous to his appearance here he had had rather high fever. The left ear-drum had been incised four days after the fever began, but was followed by only a slight discharge. Two days later a paracentesis was carried out by the attending physician on both drum membranes. However, the temperature remained between 102° and 104° F. until two days before he came to us the first time, when it was 99.6° F. per rectum in the afternoon, but rose in a few hours to 105° F.

On admission to the clinic there was a purulent discharge from both ears, especially the left one. Owing to the history of discharge for two weeks accompanied by septic temperature, namely, one in which there was great variation—as low as 99.6° F. and as high as 105° F.—it was thought highly probable that some condition complicating the mastoiditis was present. It is well known to you that in simple mastoiditis the fever, if present, is not high. It is only when some complication arises that we are likely to have either a high or intermittent fever. Furthermore, if at the end of two or two and a half weeks the amount of pus is very profuse, and of a purely purulent character without an admixture of mucus from the middle ear, together with the presence of any fever whatsoever, operation is usually indicated. We at once under ether anesthesia opened the left mastoid process and found pus in the antrum. There was, of course, very little pneumatization in this infant's mastoid. The lateral sinus was exposed and found to be gray, but there was no pus directly in the vicinity of the sinus, nor did it seem to be thrombosed. It was, therefore, not opened, but the wound merely packed and the patient returned to bed.

For a few days the baby seemed very much better; in fact, the fever was practically absent. He seemed quite bright and took his food well. Shortly after this period, however, the temperature began to show remissions, and at times would be as high as 101.2° or even 103° F., and then for a period of twenty-four or even thirty-six hours would be normal. The leukocyte count was not very high, and in view of the fact that the child

looked well, took his food nicely, and did not show the characteristic blood changes, we did not feel justified in any further exploration of the sinus or the opening of the other mastoid. At the end of three and a half weeks, when the left mastoid was fairly well healed, the temperature began to rise as high as 103° or 103.5° F. on several occasions, and in view of the profuse discharge which was then present from the right ear that side was operated. Here pus was also found in the antrum, but the sinus was not exposed. For several days the temperature was normal, when there was a sudden rise which caused great concern. However, within a few days the appearance of a measles rash showed wherein the postoperative fever had its source. Meanwhile both mastoids were dressed and the measles disappeared without any further complication. At present, as you see, the baby is entirely well, with completely healed mastoids on both sides.

In this case we had a septic temperature for several weeks before the case came under our observation, and before the first mastoid operation was performed. Nevertheless at the time of operation the lateral sinus did not show sufficient changes to warrant opening. It is highly probable that although phlebitis was present, no sinus thrombosis had occurred at any time. The somewhat irregular temperature between the first and second operation was a very puzzling feature, in that one could not be sure whether it was due to further extension of the process in the sinus of the first side operated, or whether some complication in the second mastoid was the cause. After the second operation the rise in temperature which alarmed us was due to measles. With mastoiditis even more so than with an abdominal condition it is impossible at times to know definitely what the source of temperature is. It is only by carefully weighing all the symptoms, excluding infection in other parts of the body, and using the experience gained from previous work that one can arrive at a decision regarding operation or no operation, and even then many surprises regarding pathology may be had.

Case VIII.—*Bezold Mastoiditis.*—The last case which we will present to you today is that of Mrs. J. D P., age thirty-seven,

a housewife. This patient presents a most peculiar and interesting history, all the details of which should be carefully noted. When the patient first presented herself to the clinic she gave a history that she had had for eight weeks almost continuous pain in the right ear. The physician in attendance is in principle opposed to paracentesis, and merely applied hot dressings and gave sedatives by mouth. The pains would subside after a few hours at a time, only to become more severe for several days at a stretch. For three days prior to presenting herself to us the earache had again become very severe. For two weeks before we first saw her there was considerable swelling just below the tip of the right mastoid process. This swelling had unfortunately been diagnosed by a general surgeon as a mass of enlarged cervical lymph-glands.

Examination of the patient showed a thick, red, bulging right drum membrane. The left ear was negative except for some retraction. There was redness, swelling, and slight fluctuation over the right mastoid, and this swelling extended downward below the tip of the mastoid in the region of the sternocleidomastoid muscle. The hearing for unaccentuated whispered voice was 4 meters in the left ear, but only 15 cm. in the right ear. This indicated that there was practically no hearing in the right ear, because hearing for unaccentuated whisper at a distance less than 1 meter, even though the opposite ear is tightly closed, usually means that the sounds are conducted through the bones of the skull, and transmitted to the opposite closed ear.

Owing to the condition of the patient no time was taken for an x-ray picture, since there was every indication for operation, namely, the history of long-standing pain in the ear, loss of hearing, slight fever, and the fluctuating swelling over and below the mastoid process. It is, however, a good rule that before the mastoid process is opened paracentesis of the drum membrane should be performed in the hope that it will obviate the necessity for operation, which in this case was impossible owing to the marked changes in the mastoid process and soft tissues, but, nevertheless, we incised the right drum membrane. In some cases where there is merely some tenderness of the mastoid

process, and where no paracentesis has been performed in acute otitis media, opening of the middle ear by incision of the drum membrane will often, when drainage is established, relieve the mastoid symptoms, and obviate the necessity of a mastoid operation.

In our case under ether anesthesia the right mastoid was opened. As soon as the soft tissues were entered a large amount of pus was found under the periosteum, and there was a large perforation of the mastoid cortex at the tip near its inner aspect. The mastoid process itself was completely broken down, and there was pus not merely in the cells but also in the soft tissues of the neck, to which it had escaped by way of the spontaneous perforation just mentioned. After complete exenteration of all the cells and necrotic bone the wound was packed with iodoform gauze and partly closed with several sutures, the main portion of the wound being left open.

This patient has made a most striking recovery. She was able to leave her bed five days after operation, went home in twelve days, and, as you see her today, is in perfect health. When she first presented herself you will recall that she appeared extremely ill, had lost a great deal of weight, and was very pale. She has not only regained her weight, but has been active in her work at home.

In this case we have that type of infection known as Bezold's mastoiditis which is characterized by destruction of the cortex and mastoid at the tip, usually at its inner surface, with escape of pus into the soft tissues below the sternocleidomastoid muscle. The swelling which had been diagnosed as cervical adenitis was really an abscess beneath the muscle due to the destruction of the mastoid tip.

Most authorities are agreed that paracentesis of the drum membrane is a very simple operation, rarely accompanied by any complications. Rather than wait for the pressure of pus in the middle ear to rupture the drum membrane spontaneously, it is better under gas or ether anesthesia or sometimes by the use of local anesthesia (which, however, is not very satisfactory) to incise the drum membrane. Where the latter is very thick and

resistant, the pressure in the middle ear is often not sufficient to produce spontaneous rupture; but aside from this point we believe it is much better to make an incision at the point of best drainage, namely, either in the postero-inferior or antero-inferior quadrant, than it is to have a spontaneous rupture which not only may destroy a great portion of the drum membrane and produce a permanent perforation, but may effect an opening where drainage is least advantageous. Aside from these facts, in our patient the long duration of time between the onset of the disease and the time she first came under our observation, namely, eight weeks, was more than sufficient to allow the pus in the mastoid cells to cause a marked destruction. It is fortunate for the patient that she developed the so-called Bezold's mastoiditis with perforation at the tip so that the pus could escape in the soft tissues of the neck where drainage could easily be instituted. Had this same destructive process proceeded in the direction of the lateral sinus, or middle fossa of the brain, a most serious complication, either a lateral sinus thrombosis or a meningitis, might have resulted with a very poor prognostic outlook.

While we believe that conservatism should be used in placing the indications for mastoid and other operative procedures, nevertheless there are certain surgical principles which make interference at times imperative. In an acute otitis media if there is a profuse, distinctly purulent discharge for more than two or three weeks despite all treatment; or if the pain continues, or if fever persists, and especially if it is of the intermittent septic type; or if tenderness or swelling of the mastoid process is present, then operation is indicated. In our patient, where there had been for two weeks prior to admission to the clinic a large and semifluctuating swelling at the tip of the mastoid, associated with tenderness and redness over the mastoid process itself, there was definite and urgent indication for operative interference.

CLINIC OF DR. JAMES G. CARR

Cook County Hospital

DIGITALIS DELIRIUM

THE toxic effects of digitalis have been the subject of investigation and discussion almost from the time of Withering's first paper. Those effects with which we are most familiar, the therapeutic action upon the heart and the kidneys and the unpleasant toxic action upon the stomach and bowels, were described by Withering. Indeed, his injunction was to continue the use of the drug until the appearance of symptoms referable to some one of these organs proved the effectiveness of the medication. There has been renewed interest in the subject of digitalis during the last two decades, and many workers have contributed to place our knowledge of the pharmacology of this drug upon a firmer basis and to define the indications for its use and the method thereof. The electrocardiograph has been of service in demonstrating certain features of the effect of digitalis upon the heart. With this instrument animal experimentation has been more accurately carried on, and our knowledge of the effects of the digitalis bodies has been greatly furthered. Our interest in the toxic effects of these bodies has been heightened since the publication, a few years ago, of Eggleston's work advocating the employment of much larger doses of digitalis than had hitherto been given. We are really now back at Withering's original position that the drug should be given until some definite effect thereof announces that the patient has absorbed sufficient digitalis to produce symptoms. These symptoms usually are the well-known effects upon the heart associated with the diuresis which is probably the result of the circulatory effect. A specific diuretic effect of digitalis has not been demonstrated, but symp-

toms of intoxication may appear before those announcing the
therapeutic effect.   Occasional patients will develop a headache
or a persistent nausea so early that the cardiac effects of the
drug can never be fully produced.   The larger doses which are
administered nowadays are not uncommohly followed by minor
toxic effects.   While we would not be understood as counseling
the use of ineffective doses, it is true that we do need to be on
the watch for the development of the toxic effects.

Those toxic effects which are best known have already been
mentioned.   Besides these, the disturbances of cardiac rhythm
which occur with the excessive administration of digitalis have
been fully described and the danger associated with the onset of
pulsus bigeminus has been emphasized.   In our daily work other
effects of digitalis are more or less ignored.   Quite recently
Sprague, White, and Kellogg called our attention to the eye
symptoms which are sometimes produced by digitalis.   They
state that "these disturbances are very infrequently recognized
in this country," and a little farther on, "in the French and Ger-
man literature, however, visual disorders from digitalis have
been well described."   They quote Withering, who mentions,
"confused vision, objects appearing green or yellow."   A para-
graph from their summary reads thus; "Because of the impor-
tance of noting visual disturbances in all grades of digitalis intoxi-
cation, we are reporting a series of 7 cases.   These cases present
symptoms of a toxic amblyopia with dimness of vision, flickering
and flashing scotomas, and marked disturbance of color vision.
It is apparent that visual disorders from the therapeutic use of
digitalis are more common than is supposed, and should be noted."

Digitalis delirium has been noted by a few observers, but so
infrequently that many clinicians are ready to deny the existence
of any such toxic effect of this drug.   The explanation has been
offered that such mental disturbances develop altogether upon
the basis of cerebral circultory change consequent upon the
slow pulse of digitalization, but such an explanation can hardly
hold in the face of the great number of patients who are given
digitalis until the pulse becomes abnormally slow and yet do
not become delirious.   Moreover, the slow pulse of heart-block,

which may last for years, is compatible with normal mental vigor. Duroziez published a list of case reports in 1874, and in 1901 and 1905 Hall published 2 cases. These reports will not stand rigid criticism. Some two years ago Hamburger published in these Clinics some observations upon this subject, and I want now to direct your attention to 2 patients.

G. K., a man of fifty-four, was admitted to the hospital May 31, 1925. He gave a history of dyspnea and intermittent precordial distress lasting over two years. Recently his legs have been swollen at night. He had syphilis twenty years ago. Physical examination shows an enormously enlarged heart, mainly to the left and downward. The apex-beat is in the midaxillary line. At the base there is a marked systolic murmur followed by a louder diastolic murmur. There is also a systolic murmur at the apex. The Corrigan pulse, the capillary pulse, and the Duroziez sign are all present. The lower edge of the liver can be felt and is tender to pressure. The feet and legs are markedly edematous. The blood-pressure is 194/72. The diagnosis is syphilitic aortitis, aortic regurgitation, cardiac hypertrophy and dilatation, relative mitral insufficiency, and hypertension with cardiac decompensation. The Wassermann reaction was strongly positive. The patient was given potassium iodid and 15 minims of tincture of digitalis; the latter was given four times a day. This was kept up from May 31st until June 6th. At this time mercurial inunctions were begun. Commencing June 18th digitalis was again administered, this time in the form of the powdered leaf, of which 1 grain was given three times a day; this was continued until July 3d, a total of eighteen days, with the administration of 54 grains of powdered digitalis leaf. Beginning July 4th the tincture was given again in 20-minim doses three times a day. After the 16th the pulse was below 64, upon one occasion being recorded as low as 44. On the 18th the dose was halved. On the 20th the nurse's record contains this statement, "Talk is irrational, will not stay in bed, talkative, and the talk is irrelevant." The digitalis was stopped. He had been given digitalis continuously for thirty-six days. As already stated, he was given 54 grains of the powdered leaf in eighteen days; this was

followed by 16 drams of the tincture in a second period of eigh-
teen days. At the end of this latter period he was delirious. The
delirium was not preceded by headache or nausea. Several
records were made referring to an arhythmia, but, unfortunately,
no electrocardiograms were obtained. The cardiac condition
and the decompensation were practically unchanged as com-
pared with the time of admission. In spite of this intensive
digitalis therapy and the prolonged use of mercury and po-
tassium iodid, the patient's general condition is now worse than
it was in June. The prognosis is correspondingly bad.

After the digitalis was withdrawn, without any other change
of the medication or general treatment, the delirium disap-
peared. Seven days thereafter he was returned to his old place
in the large ward (he had been isolated because of his noisiness),
though at times he still talked at random. He was given no
more digitalis until August 1st, following which date he took 1
grain of the powdered leaf three times a day for eight days.
Some signs of returning delirium led to the prompt withdrawal
of the drug. After two days he was again given the tincture in
doses of 15 minims three times a day, which he has now taken
for three days without disturbance.

The second patient is an elderly man who has been about
the hospital for years. He is, in fact, a resident of the hospital
rather than a patient, though he came here originally for broken
compensation. Indeed, he has not been free from fairly well-
marked edema of the legs for years and is short of breath upon
slight exertion, yet he has worked here in the Roentgenological
Department. About two or three times every year he spends a
fortnight or so in our ward; while compensation is never re-
stored, the rest is good for him. He goes back to his work with
an improvement of his cardiac function at least. One week ago,
August 5th, he was sent to our ward because of his queer con-
duct. He had put the records in his desk in disorder and could
give no reason for his actions; indeed, he did not seem aware
that he had done anything unusual. He did not seem to know
at all what he was about, and had been guilty of many obviously
peculiar actions. After he was brought to our ward he was put

to bed, much against his will. He was unruly, irritable, and restless, though his fellow-workers said that he had been inclined to drowsiness for two days. In the ward he would not eat, he refused any co-operation with the nurses, obeying no rules, and following no instructions. He would talk very little and wanted to be left alone. He was especially confused about his whereabouts, did not seem to know where he was, and was watching every opportunity to get up and start for the *x*-Ray Department. This attitude was most unusual for the patient, who was normally a genial man, one who has always been everybody's friend, and much less easily irritated than the average man. Not long after reaching the ward he left his bed and with very scanty clothing started for his place of work. This irrational, disoriented condition lasted for forty-eight hours after he became formally a patient. During this time his heart rate was 44 to 48. He awakened on the third morning with a marked change; the delirium was apparently gone, though he was not yet quite recovered, as would appear when he attempted a conversation.

With this return of his mental faculties we were able to get a connected history of his symptoms. In the absence of any other demonstrable cause for his delirium we had surmised that digitalis might be the responsible factor, for we knew that long-continued use of the drug largely upon his own initiative had given him confidence about its use which might have led him into taking enough to produce toxic symptoms. As he recovered he told us that for some time before he was taken sick he had been taking three 2-grain capsules of powdered digitalis a day. He was unable to say over how long a period this had continued. Three days before coming under our care he was drowsy and apathetic. On the day before any strange actions were noted he was nauseated and vomited. He had no diarrhea—which in our experience is a rare symptom of digitalis intoxication—until two days after he became a patient. A peculiarly interesting feature of his case, especially so in the light of the reference already made to the work of Sprague, White, and Kellogg, was his statement that for three weeks before the onset of other symptoms

he had noticed a gradually increasing blurring of vision. This disturbance of vision was still so pronounced after the disappearance of his delirium that for a few days he was entirely unable to read. He is slowly regaining his sight.

In connection with this particular symptom there comes to mind a man whom I had under my care last winter. This patient was sixty-six years of age and suffering more or less chronic distress of a minor degree from decompensation. From time to time he had taken small doses of digitalis for fairly long periods. After a while it became necessary to increase the dose, and after taking the larger dose for a few days he called me up one day to tell me that he was losing his eyesight. He attributed the visual disturbance to the medicine, but I assured him that I did not believe it possible that digitalis would produce such symptoms, and asked him to see an ophthalmologist. He saw not only one, but several; no disease of the eye or the optic nerve could be discovered, and finally it was concluded that the patient must have had some disturbance of the visual center the result of vascular disease. He was given potassium iodid and slowly recovered. The digitalis for a time was stopped, and when started was administered in small doses. Some three months later, after he had pretty well forgotten the experience, it was deemed advisable to again increase the dose of digitalis. I was not only surprised but informed when he called up within a few days to tell me that the old visual disturbance was coming on. In spite of my original belief that the digitalis could not be the cause of the visual disturbance, I am now satisfied that such was the case.

We believe these 2 patients have suffered from a delirium due to the digitalis. The second case is especially convincing. A patient who had taken digitalis for some time developed a delirium without the presence of any disease which might be regarded as the cause of the mental disturbance, the delirium was preceded by failure of vision and by nausea and vomiting, and was associated with a pronounced slowing of the cardiac rate; a diarrhea began two days after his admission as a patient; finally the delirium disappeared two days after the known withdrawal of the digitalis. It is likely that the patient's mental

state during the two days immediately preceding his coming under observation was such that he neglected to take his medicine; about that no one could be sure. At the end of a week his mind is perfectly clear. The second case is more open to objection because of the presence of a positive Wassermann reaction with sluggish ocular and patellar reflexes, and also because of the long-standing decompensation with no improvement. Yet, as we assemble the facts, the delirium can hardly be attributed to anything but digitalis intoxication. He was delirious only for a few days; the delirium followed the administration of digitalis for a period of thirty-six days and subsided about six days after the drug was stopped. The degree of decompensation was not perceptibly altered for weeks before the onset of the mental upset, nor did the decompensation subside as the delirium disappeared. The short duration of the delirium is likewise evidence against the probability of syphilis as the etiologic factor. With the increase of the dosage of digitalis a week later minor symptoms developed, but subsided with the withdrawal of the drug. The slow pulse, much slower just before the onset of delirium than at any time before or since, may be regarded as a manifestation of the digitalis effect and strengthens our belief that the delirium was the result of digitalis intoxication.

The diagnosis of digitalis delirium must be made with reserve. Decompensation probably with the associated derangement of cerebral circulation is a more common cause of delirium than is digitalis. The mere development of delirium during the course of the administration of digitalis is not sufficient justification for the diagnosis of digitalis toxemia. We have been interested in the subject for several years and have been on the watch for cases, yet only a few well-established instances have been seen, and often the delirium which has called our attention to the possibility of digitalis intoxication has been shown to be due to some other cause. I recall a Mexican whom we had on our service some years ago. This man had a serous tuberculosis which particularly involved the pericardium, and in the course of his pericarditis he developed an edema, for which he was

given digitalis. This was continued for some time. During the digitalis medication mental disturbance was noticed. No one in the ward could speak to the patient, so that an exact idea of his aberration was not to be obtained. Like the 2 patients here presented, he changed from a quiet, tractable patient to one who was irritable, unruly, and inclined to make trouble. The digitalis was withdrawn and almost simultaneously there was definite improvement in the patient's mental state. However, the symptoms of mental derangement reappeared before digitalis was again administered, so that the medication was obviously not responsible. Henceforth the mild delirium was present, to a greater or less extent, throughout the rest of the patient's life, without any reference to the administration of digitalis. At autopsy there was found a tuberculoma of one frontal lobe.

The diagnosis of delirium due to digitalis rests upon: (1) the administration of the drug in rather large quantities or over a long time (we have not seen it develop early in the course of digitalis medication unless the drug was used in large doses); (2) the absence of any other probable cause for the delirium; (3) the presence of other symptoms of digitalis intoxication; (4) the onset of the delirium as the decompensation recedes; (5) the rapid clearing of the mental state after the digitalis is withdrawn. Though digitalis delirium is infrequent, it is important. If this condition is ignored the delirium is apt to be ascribed to the cardiac disease, and the drug pushed, to the great disadvantage of the patient. Wherever digitalis is used over any length of time it should not be forgotten that the onset of any mental disturbance calls for a careful consideration as to the possibility that digitalis has excited the mental symptoms.

A CASE OF UREMIA

Our next patient is a man of fifty-nine, a tailor by trade. He is married and his wife and 2 children are living and well. He gave the following history: For ten years he has been nervous and for the past six months he has noticed pain in both shoulders. He also states that for the past two years he has had pain in both loins. This pain is constantly present, is localized to a small area, and never radiates. It is of a dull aching character, worse during the day. The pain in the shoulders is excited by movement. This has been present for six months and is gradually growing worse. In the last six months he has lost 20 pounds in spite of a fair appetite. His bowels are regular. He has had occasional vomiting spells, about which no more accurate information can be obtained. There is slight dyspnea upon exertion. He has no swelling of the feet and no cough. He gets up some four or five times a night to urinate. He has occipital headaches at times and occasional attacks of dizziness. He denies any other illness at any time. He uses no alcoholic drinks, but does use tobacco, tea, and coffee.

Physical examination revealed a fairly well-nourished elderly man who did not appear acutely ill and did not regard himself as sick. The pulse-rate was 80, temperature 98.6° F., and respirations 22. The blood-pressure was 230/120. There were several bad teeth. The pupils reacted normally. The thyroid showed a general enlargement. The lungs were normal. The apex-beat was located just outside the nipple line in the fifth interspace. The sounds were distant. A systolic murmur was heard at the apex and was transmitted to the axilla. The second sound over the aortic area was accentuated. Aside from slight tenderness upon palpation of the liver the abdominal examination was negative. The prostate was found to be moderately enlarged. The extremities were negative except for a fine tremor of the hands and fingers. There was bilateral tenderness over the posterior superior iliac spines. The urine contained albumin

in rather large amount.   The hemoglobin was 60 per cent., erythrocytes 3,440,000, leukocytes 11,600.   The Wassermann was negative.   Three days after admission the blood urea was 39.98 mgm. per 100 c.c. of blood, uric acid 3.75, and creatinin 1.50.   The basal metabolic rate was 60 per cent. plus.   Since his admission to the hospital the daily output of urine has been less than 500 c.c., with one exception.   Until these examinations were completed the patient was not kept in bed.   Since the demonstration of the high basal metabolic rate he has been so confined.   After he had remained in bed two days the blood-pressure was found to be 140/80.   This was confirmed by two examinations upon subsequent days.   Albumin has been present in the urine at every examination.

Upon the twelfth day of his stay in the hospital a second specimen of blood was sent to the laboratory for examination. The report differed so widely from the one just quoted that another sample was sent for confirmation.   The house physician was certain that some mistake had been made.   The results of the examination of the third specimen corresponded closely to the second.   There had developed in the nine days elapsing between the first examination and the two subsequent ones a surprisingly high retention of the non-protein nitrogenous constituents of the blood.   The urea at this time was 229 mgm. per 100 c.c. of blood, uric acid 5.20, and creatinin 5.85.   The patient's clinical condition was practically unchanged.   He had been regarded as doing well until the return of this report with its evidence of advanced renal insufficiency.

Yesterday in the afternoon (the fourteenth day after admission) the nurse made a record thus, "Condition good." Within five hours another record was made, "Patient having convulsion."   Over a period of an hour or more the patient had several convulsions and throughout the most of the night he was in a very serious condition.   Fortunately, after several hours of vigorous treatment, the patient has responded and is much better this morning.   Now he is rational, comfortable, and decidedly improved.   The pulse is 99, respirations 30, and the temperature 100° F.

Particular interest attaches to the case because of the sudden change from a state which aroused no concern to one in which the patient was manifestly in a very serious condition. This change was not entirely unforseen. After the examination of the blood had revealed advanced renal failure with extreme retention of the nitrogenous bodies, the convulsion was an event which caused no surprise. It is clear enough that we might have been astonished by the rapid appearance of the uremic state, except for the prognositic information derived from the blood examination. The course of this case makes it easy to appreciate why, now and then, uremia appears with dramatically sudden onset in an individual who has not been regarded as actually sick. This man came to the hospital for diagnosis rather than for treatment. He had not been quite well, and his friends persuaded him to enter the hospital that he might have a complete examination. Except for the presence of the high basal metabolic rate and the tremor, nervousness, and loss of weight in spite of a fair appetite it is probable that he would have been about the ward when the convulsion set in. Perhaps he would have been allowed to go home as he desired after the examinations were finished. In either event the convulsion would have come out of a clear sky. Uremia may cause sudden death. In our teaching we are apt to utter warnings lest uremia be diagnosed too frequently, and hence, incorrectly, in the presence of convulsions or coma in a patient who is found to have an albuminuria. Our experience teaches us that frequently too much importance is attached to the finding of albumin in cases of coma, with the result that too often uremia is diagnosed and incorrect treatment is carried out. We need to lay stress upon the danger of mistakes when diagnoses are hastily made in the presence of these outstanding symptoms. It is right to emphasize these facts and to insist upon careful consideration of other symptoms and physical findings. The character of the attack and its mode of onset and the previous history of the patient are also of importance. Yet, in our zeal to avoid too ready acceptance of uremia as a diagnosis, it must not be ignored or forgotten that uremic coma, with or without convulsions, may develop

quickly in an individual apparently well and may lead to death within a few hours. The coma is not invariably accompanied or preceded by convulsions. Not infrequently the comatose state is preceded by a short period of mental confusion or by headache and vomiting.

Our experience with this patient illustrates very well the difficulty of prognosis in the presence of an established nephritis. Even those cases which have been most thoroughly worked up are apt to defy any dogmatic rules of prognosis. In this case the sudden change for the worse, so marked in degree, in the results of the chemical examination of the blood indicated the onset of serious symptoms. Yet we have seen cases in our own ward in which similar high values of the nitrogenous end-products have not been followed shortly by an outbreak of uremia. I recall the case of a man whom we had in the ward some three years ago. This patient came to the hospital because of shortness of breath. The intern found his blood-pressure high and sent a specimen of the blood for examination. The first report showed values so exceptionally high that a second specimen was sent at once, in the belief that some mistake had occurred. The presence of some 6 mgm. of creatinin per 100 c.c. of blood, in association with proportionately high figures for the other non-protein nitrogenous bodies, was confirmed. This patient was in the ward for five months; the values for creatinin were never below 6 mgm. per 100 c.c. of blood, and were often higher. In spite of several attacks of impending uremia (the patient never did go into coma nor did he have a convulsion), he lived in the ward for some five months and was finally discharged to the County Poor Farm in fairly good clinical condition. Admittedly such a case is most unusual.

Our knowledge of nephritis in all of its aspects is still so far from complete that consideration of these extremely unusual conditions serves to emphasize to us the importance of care in our statements regarding prognosis. Hardly any subject in medicine demands of us more caution. Even though we may feel that a given case has been thoroughly worked up, both from the clinical and the laboratory aspects, it is well not to make our

prognoses without careful thought about the qualifications that may be in order. Not long since, in the course of a few months, 3 patients in our ward, each of whom had at the time of the outbreak values for the non-protein nitrogenous bodies about equal to those shown by this patient at the time of his admission, were abruptly taken with convulsions which we could only regard as uremic. On the other hand, high values for these substances are compatible with improvement and the patients so afflicted are not always doomed to death within a few days or weeks. One principal difficulty in making prognosis is due to the variable response to treatment shown by different patients. There is now a patient on our service who had upon his entrance, two months ago, values for these non-protein constituents almost three times as great as those of this patient at the first examination. This first man has shown a splendid response to treatment, is better clinically, and the blood examinations reveal less than half the former grade of retention. Too much reliance should not be laid upon any single phase of the nephritic manifestations in rendering a judgment as to the patient's outlook.

In this particular case clinical findings and laboratory results alike point to the presence of an advanced nephritis with pronounced failure of renal function. The events of the last few hours are impressive, especially in the light of the rapid aggravation of retention during the past twelve days. Here is a man who has probably had a progressive disease of the kidneys for years, who comes under observation in apparently good condition, certainly not worse than some patients who pass through the ward every year without untoward symptoms, yet within a few days renal function tests have shown marked impairment, and shortly thereafter the patient goes into uremia, with convulsions and coma so severe that his life is despaired of. The onset of uremia quickly following the demonstration of rapidly increasing nitrogenous retention signifies sudden and severe renal insufficiency. It is reasonable to surmise that similar abrupt changes in renal function may be responsible in many of these cases of nephritis for uremia which comes on very rapidly.

Thus far we can learn from this case two important facts regarding the course of chronic nephritis: (1) chronic nephritis is often an insidious disease running into an advanced stage with few or minor symptoms; (2) prognosis is difficult, a matter of judgment, not of precise demonstration. The words of Hippocrates come back to us as Osler quoted them, "Experience is fallacious and judgment difficult." The abrupt appearance of the uremic convulsion with the consequent danger to the patient's life, though fortunately he has improved, warrants us in emphasizing another feature of uremia of which we have spoken. Uremia may come on very quickly and cause death within a few hours of the initial symptoms.

We can offer no explanation for the exacerbation of symptoms nor for the rapidly progressive diminution of renal function. The daily records of the senior intern indicate that he was satisfied with the patient's condition and that nothing had occurred to the patient which would lead him to anticipate an acute exacerbation of the renal disease. From the clinical standpoint there was no warning of the serious failure of renal function; the laboratory evidence of these changes came as a surprise. The patient who had been at rest in bed, on a careful diet, shortly after the discovery of the severe retention goes abruptly into a uremic convulsion. It is not uncommon to see the uremic state develop rather quickly after an acute nephritis has further impaired the functional efficiency of kidneys involved in an old process. The acute process in such a case is usually the result of some fresh infection, which may be of minor importance in itself. With one qualification this man has had no infection and has been protected so far as possible from the opportunity to acquire an infection. The exception was this: several bad teeth were removed. Before the extraction he had been in the hospital five days, during which time his temperature had gone to 99.2° F. once; no other reading had been above 99° F. For four days following the removal of the teeth his temperature went to at least 99.4° F. once a day; upon one occasion the temperature was 100° F. There have been, however, no changes in the urine which would justify the diagnosis of an acute exacerbation of an

acute nephritis. We have no satisfactory explanation to offer for the outbreak of uremia. In the wards of the County Hospital we meet many patients with infections about the teeth. In such cases extraction is often advised not as a curative measure for existent pathology elsewhere, but as one justified by general principles of prophylaxis. I doubt if such practice has proved its value in nephritics. Many such patients have been in the wards for long periods of time after the extraction, yet I have not seen the course of a nephritis apparently altered as the result of the operation.

After the diagnosis of nephritis complicated by hyperthyroidism was made the patient was kept in bed and given a diet with a protein maximum of 35 grams per day. The salt was restricted. After the discovery of the rapid accumulation in the blood of the non-protein nitrogenous bodies, 800 c.c. of Fischer's solution was injected intravenously, and upon the next day this therapy was repeated after the withdrawal of 100 c.c. of blood. On the day following the first of these injections there was an increase of the urinary output to 1000 c.c., twice the usual daily output heretofore. This response was not met with the second day. After the convulsion yesterday 600 c.c. of blood were withdrawn and a third intravenous injection was given. While we believe venesection to be one of our most valuable therapeutic procedures in impending or established uremia, we are not likely to continue the treatment in this case. You will recall that his erythrocyte count upon admission was 3,440,000; more than a pint of blood has been taken from him since that count was made. One of the outstanding features of nephritis is a tendency to anemia. The withdrawal of blood from a nephritic with an established anemia means the aggravation of a condition which the patient has already shown his inability to overcome. It is our practice not to bleed an individual whose red count is below 3,000,000. Indeed, we hesitate to advise venesection if the red count is as low as 3,500,000. In the presence of such figures and the presence of uremia the decision as to the use of bleeding must depend upon individual indications.

After the convulsions ceased the patient was sweated. Our experience has convinced us of the favorable effects of sweating, though the desired results are not constantly obtained. The patients with general anasarca are likely to sweat poorly. The presence of symptoms of cardiac weakness should warn us to be careful in our endeavors to produce sweating; the depressing effect of an artificial sweat may prove disastrous. There is, finally, a borderline group of patients who do respond to the sweating process, but this is accompanied with a disproportionate amount of depression. In such instances the continuance of the sweats is a matter for individual judgment.

Twenty c.c. of spinal fluid were withdrawn yesterday. The fluid was under a slightly increased pressure. Spinal puncture has been employed with the purpose of relieving intracranial pressure upon the theory that uremia may be the result of cerebral edema. This patient's condition did not appear to be changed by the puncture. The fluid was sent to the laboratory for examination and the interesting observation was made that the spinal fluid contained 157.38 mgm. of urea per 100 c.c. of fluid.

Nothing has been said about the possible relationship of the uremia and the hyperthyroidism, because there is no reason for connecting the two conditions etiologically. In the light of anything we know at present, we must be content to regard the hyperthyroidism as a complication of an old nephritis. Neither have we discussed the marked fall of blood-pressure which occurred after the patient was put to bed. Unfortunately, daily readings of the blood-pressure have not been taken, so we are not in a position to discuss any possible relationship of the lowered blood-pressure to the onset of the uremia. There is no justification for discussing the subject here further than to suggest that the abrupt fall of pressure might have been associated with so marked a change in the mechanism of urinary secretion of a kidney accustomed to higher levels of pressure as to have been partly responsible for the rapid accumulation of nitrogenous end-products in the blood and the onset of the uremia. Usually, however, we have found that just prior to the onset of

uremia the pressure is apt to be rising, sometimes quite markedly. In this case no reading was obtained for several days before the convulsion.

Further course of the case: On the third day after the first convulsion another occurred. At this time the blood-pressure was 240/160. He was much better the following day. He took his food with relish, even sitting on the side of his bed to take his meals. Toward evening he became drowsy and gradually sank into a coma from which he did not rouse.

At autopsy a chronic glomerular nephritis, small granular kidney of advanced degree, was found. There was hypertrophy of the left ventricle and generalized arteriosclerosis. An old abscess of the prostate was found; doubtless, the prostatic condition was largely responsible for the nocturia of which the patient complained when he came to the hospital; perhaps for the pains in the loins mentioned in the history. The futility of extraction of teeth while suppuration exists elsewhere needs no emphasis.

HYPERSENSITIVENESS TO MILK COMPLICATING THE TREATMENT OF DUODENAL ULCER

YOUR attention is asked to the next patient that we may briefly discuss an unusual complication which occurred in the course of the dietetic management of a duodenal ulcer. This man, thirty-two years of age, was admitted to the hospital June 6, 1925. He stated that he had suffered from "stomach trouble" for the past year or more. He had dull gnawing pains in the epigastrium which came on about one to three hours after his meals. Food and hot drinks would relieve the pain. He did not pay much attention to these symptoms and did not consult a physician. About two weeks ago, while riding on a street car, he was suddenly taken with an attack of dizziness and weakness. He fainted and was taken home. Since then he has been in bed. He is comfortable while quiet, but becomes dizzy if he attempts to sit up. During these two weeks his stools have practically all been tarry. He has not had nausea, vomiting of food or of blood. Since the attack described he has not suffered with the old abdominal pain. His appetite has been good and food has not excited the pain from which he formerly suffered. There is no history of previous illness. He denies venereal disease.

Upon examination the most striking finding was the marked pallor of the patient. The conjunctivæ and the oral mucous membrane shared in the general appearance of anemia. The pulse was rapid, ranging from 110 to 120, and there was an afternoon rise of temperature to a trifle above 100° F. The cardiac borders were normal; a blowing systolic murmur was heard at the apex. The lungs were normal. Abdominal examination was negative. There was no tenderness, no rigidity, no distention, and no masses were felt. Rectal examination revealed the presence of a small amount of tarry fecal matter, which gave a strongly positive reaction for blood. The extremities were negative. The reflexes were normal. The blood-count showed reds

1,740,000, hemoglobin 30 per cent.; blood-pressure 120/84. The diagnosis of bleeding ulcer of the duodenum was made, morphin was given, an ice-bag was applied to the abdomen, and 30 c.c. of horse-serum were given intramuscularly. On the day following admission Sippy management was begun; he was given small quantities of milk and cream every hour, followed on the half-hour by an alkaline powder. On the 8th he complained of headache, which the record says was "severe"; on the 9th and 10th the record reads that he "still complains of headache." On the 11th he complained that the milk nauseated him and the headache persisted; in addition to these symptoms he was quite drowsy all the next day. On the 13th an urticaria appeared quite suddenly, widely distributed over the body. The temperature rose to 101.2° F. and he vomited a large amount of soured milk. He insisted that he had never been able to take any considerable quantities of milk without unpleasant symptoms. The milk was stopped and another diet arranged. Nevertheless the temperature went still higher upon the following day, reaching 102.4° F., and with this highest temperature there was pain and tenderness in many of the joints. These joint pains lasted through another day, as did some minor manifestations of urticaria. On the third day after the milk was stopped the temperature fell to more nearly normal than at any time since his admission, the urticaria and joint pains disappeared, the headache was gone, and the patient was less drowsy than for several days.

With the fundamental disease in this case I do not propose to detain you, neither do I mean to discuss the hemorrhage and its treatment. I merely want to note the presence of this uncommon complication of the usual dietetic management, a definite protein reaction to the milk. We see many cases of peptic ulcer in these wards, perhaps in a single ward 40 or 50 a year, so that one's experience here and in private and dispensary practice easily runs into hundreds of cases in the course of a few years. This is the only case of such definite and unquestionable sensitization to milk which I have seen in the use of this diet. An effort was made to determine whether or not the casein or lact-

albumin was to blame, but to neither of these was there a satisfactory reaction. So far as the treatment is concerned, we did nothing but restrict the excessive use of milk, and the symptoms disappeared. Instead of compelling the patient to take nothing but milk, he was allowed cereals, soft eggs and puddings, and milk toast. Milk was given with his cereal. With these comparatively small amounts of milk there was no recurrence of the reaction and, as you can see, he has made a steady gain and will soon be able to leave the hospital.

CLINIC OF DR. JESSE R. GERSTLEY

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL AND MICHAEL REESE
HOSPITAL

EMPYEMA OF UNUSUAL ORIGIN

BEFORE demonstrating this specimen I should like to describe a most remarkable experience:

A little girl, aged fourteen years, complained of some difficulty in swallowing. Insignificant at first it gradually grew more severe. After some weeks she described a feeling of pressure behind the sternum. Food after being swallowed would give a sensation of sticking in this region. At first there was little pain, but later some pain was associated with swallowing, being referred to the substernal region. At other times pain was absent. As the pain became more marked she learned that it could be alleviated by taking fluid food, and so, of her own accord, she gradually selected a liquid diet.

Physical examination showed the little girl in good condition, but slightly undernourished. There were no findings whatsoever: nor did the blood, urine, or Pirquet test give any clew. Fluoroscopic and x-ray examinations with the use of barium showed the swallowed semisolid medium pass rapidly down to just above the cardia. Here was some difficulty, the lumen of the esophagus becoming quite narrow for a distance of about 1 inch. The borders of this narrowing were smooth and round.

In consultation with a gastro-enterologist of great experience, her physician agreed to the use of the esophagoscope. The operator passed the tube very gently and carefully, meeting no resistance whatsoever until it reached a distance of a few inches above the cardia. Here he felt just a tiny bit of resistance which immediately melted away. The appearance of the mucous

membrane of the esophagus was normal in all parts except in this area, where for a space of about 1 square inch the mucosa on the posterior wall seemed eroded and allowed a slight oozing of blood. Immediately following the withdrawal of the tube the child experienced terrific pain in the same region, so severe that she almost fainted and had to lie down. After about one hour she felt a little better and went home. In her own home the pain became severe during the night. Fever and a new distressing pain in the left chest made her most miserable.

In the morning, when I saw her for the first time, she presented a most striking and puzzling picture. Cyanotic and in collapse, she was almost moribund. Her temperature was between 100° and 101° F.; pulse rapid and weak; respirations rapid and accompanied by an expiratory grunt. Pain in the left chest was excruciating. The whole physical and mental condition of the patient resembled one of those virulent and fatal empyemas which were so terrible in the recent Army Medical Service. That an empyema could have developed, however, seemed impossible. We first thought of the possiblity of hemorrhage and shock in view of the relatively low temperature. The blood-count, however, showed a normal red count and hemoglobin and a leukocytosis of 18,000. The patient was altogether too sick to be moved, and a portable x-ray machine was employed, but the plates were of no value.

Shortly physical findings in the left chest rapidly assumed the flatness of an empyema. Aspiration showed a large quantity of serosanguineous fluid. The patient was in extreme discomfort and life was sustained by injections of morphin, stimulants, and constant inhalations of oxygen. Following the aspiration the patient was relieved for a few hours, but rapidly became worse. She now refused even liquids, and, in view of a possible rupture of the esophagus into the pleura, we were afraid even to attempt feeding by gavage. At this time the gastro-enterologist attempted to pass the finest sort of tube, but some obstruction to its passage foiled his attempt. There was no vomiting of blood, nor was there any blood in the stool. After a few hours the patient lapsed into semiconsciousness and died shortly afterward.

The whole affair was so rapid, so virulent, and so puzzling that we exerted ourselves to the utmost to obtain postmortem examination, and we were able to obtain permission for a section of the chest.

The cause of the entire trouble is the hazelnut size tumor which I have in my hands. This lay in the posterior wall of the esophagus. The cavity of the tumor was filled with purulent material, the walls being of fibrous tissue. Our pathologist reports some nerve tissue also present, which may place the neoplasm in the class of dermoids. The wall of the esophagus was unbroken. The wall of the tumor, however, had ruptured, and the purulent contents discharged into the pleural cavity. The whole picture resulted from the pressure of the esophagoscope.

There is no question in my mind that this tumor would have ruptured spontaneously. The esophagoscope was passed most skilfully and the resistance was so slight as to be almost negligible.

There were no other pathologic findings except some serosanguineous fluid in the left pleural cavity. No pneumonia was present.
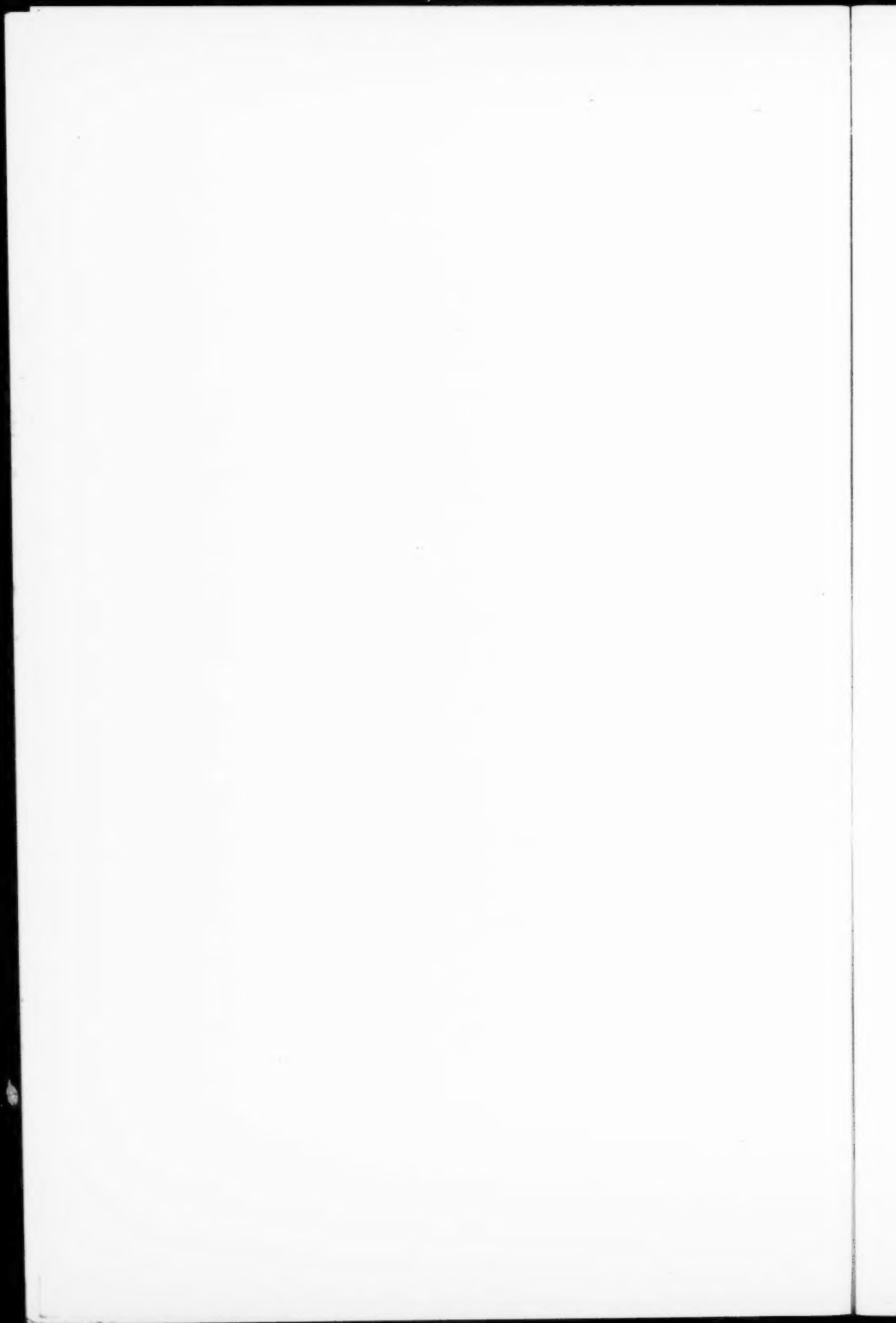
SUDDEN DEATH FOLLOWING PROPHYLACTIC INOCU-
LATIONS AGAINST DIPHTHERIA

THIS seven-year-old girl lying in coma presents an interest-
ing problem of diagnosis. As you see, she is quite irrational,
tossing in bed, crying occasionally as if in pain, and making aim-
less movements of the hands and legs. Her general nutrition is
good. There is no sign of any external injury. Her tempera-
ture is 103° F., pulse 116, respirations 28, blood-pressure 120
systolic and 75 diastolic.

The skin is warm to the touch and, other than an erythema
of the face, shows no sign of inflammation.

Looking at her more carefully we find an internal strabismus.
Her pupils are widely dilated and react neither to light nor ac-
commodation. The pharynx is pale and the tonsils small. Her
irrational condition prevents any conclusion as to the deviation
of the tongue.

Her neck shows some rigidity, and note that when I bend
her head upon the thorax both her thighs are markedly flexed
over the abdomen. The Brudzinski sign, then, is also clearly
positive.

In the thorax there is neither dulness, râles, nor abnormal
breathing. The heart is not enlarged. The sounds are clear
and regular, though quite rapid. The abdomen is retracted, but
shows no rigidity nor tenderness, and no enlarged organs are
to be felt. The abdominal reflexes are absent.

Upon inspection the extremities appear normal, but notice
that it is difficult to extend the leg when the thigh is flexed on
the abdomen. The Kernig sign, then, is positive. This is in ac-
cord with the Brudzinski noted a minute ago.

As to the laboratory findings, the blood examination shows
50 per cent. hemoglobin, 2,000,000 red cells, 26,000 leukocytes.
The differential count gives 47 neutrophils, 52 small mononu-
clears, 1 large mononuclear. The von Pirquet test is negative.

In a child showing symptoms of this sort lumbar puncture is, of course, urgently indicated. The fluid came out absolutely clear under normal pressure, with no albumin or globulin, with a negative Lange, and with only 8 cells per cubic centimeter. The urine is surprising; the color is bright red, it is loaded with albumin, and microscopic examination shows innumerable red cells. The blood chemistry gives a non-protein nitrogen of 410 with 7.1 creatinin.

The history of this little girl is of great interest and is very confusing. According to the mother, the child was in good health until about six days ago. At that time a rash appeared upon her face and she started to vomit. She felt very tired and weak during these six days. The vomiting lasted for two days and the stupor and coma began yesterday. There is nothing else of any importance. The only other bit of information elicited is that the child had the third inoculation of T and A mixture as a prophylactic against diphtheria two days before the onset of the symptoms.

We have here a real problem in diagnosis. The onset of this illness immediately following the use of the T and A would, of course, suggest that mixture as the etiologic factor. However, that combination has been used in so many thousands and thousands of cases with no bad results that I would hesitate to ascribe the symptoms to it until every other possibility had been ruled out. We must consider meningitis, but normal spinal fluid in a condition of six days' duration would practically rule out such possibility. That most of the symptoms may be ascribed to uremia cannot be questioned. But what is the etiology of the uremia? The history of a rash would strongly suggest scarlet, with a secondary nephritis. However, there is no history of a rash any place other than on the face. Second, a typical scarlet nephritis rarely occurs as early as this.

Again, we have strabismus. This might occur in uremia with localized cerebral edema, but, on the other hand, it certainly strongly suggests some intracranial pathology. Possibly we have an encephalitis complicating a nephritis.

You see I am doing everything possible to avoid a hasty

superficial indictment of the T and A mixture.   At any rate the solution of the question is not absolutely clear.

Subsequent Course.—*Two Days Later.*—I have here the pathologic specimens of the little girl shown previously.   Immediately following the last clinic she became worse.   A venesection was performed and a blood transfusion of 200 c.c. from the father was given.   Following this the pulse and respiration improved, though the coma was unaffected.   To our astonishment, some hours later symptoms of hemiplegia developed, with paralysis of the muscles of the left face and twitchings of those of the right arm and leg.   Lumbar puncture was performed twice following the onset of the hemiplegia, and in each case the findings were absolutely normal, with the exception of a few red cells in the spinal fluid.   The coma remained as before.   Last night the pulse and respirations decreased gradually and finally became imperceptible.

Fortunately we were able to obtain a postmorten examination, and I will submit a brief summary of the detailed examination made by our pathologist, Dr. O. T. Schultz.

The color of the skin was yellowish brown.   Numerous bluish-black areas of discoloration varying in size from 0.5 to 2 cm. in diameter were scattered over the arms, forehead, and lower extremities.   There was a hemorrhagic excoriation of the inner surfaces of the thighs just beneath the perineum.

There was generalized lymphadenopathy.   As regards the head, we were surprised, upon removing the calvarium, to notice that the brain was smaller than usual and filled the cranial cavity incompletely.   Shining through the left portion of the dura was a bluish-black mass which was slightly movable.   On removing the dura this became apparent as a non-adherent, rather firm blood-clot which appeared to be of recent duration, covering much of the left hemisphere.   There were no other findings in the brain and nothing unusual in the chest and abdomen.

Considering the organs individually, the lungs showed no abnormalities.   The thymus measured 2 by 1.5 by 0.5 cm. and was pinkish yellow.

The heart gave us a surprise in showing some fatty change in the muscle-fibers. It was quite apparent that this fat was of the muscle itself and not extraneous tissue.

The spleen was quite small and firm. The liver showed no gross change, nor did the gastro-intestinal tract, the adrenals, or pancreas. The kidneys, however, were clearly diseased. The capsule could be removed from each without difficulty, disclosing a reddish-brown organ, the surface of which was dotted with numerous pinpoint, often confluent, bright red areas. On section the glomeruli were seen as glistening, bulging, bright red dots against the brownish-yellow parenchyma. Scattered through the cortex and pyramids were long yellowish-white streaks which were evidently fatty in character. The organ bulged somewhat on section.

The detailed and painstaking microscopic examination confirmed the gross examination, namely, acute hemorrhagic glomerular nephritis, acute interstitial myocarditis, subdural hemorrhage, ecchymoses in the skin and beneath the dura, edema of the lungs, dehydration, and anemia.

We still have not solved this problem. Encephalitis is certainly ruled out. Whether the child had a nephritis and the inoculations of the T and A mixture precipitated all the trouble is, of course, a possibility. That the trouble was due primarily to the T and A mixture I am loath to admit, and still we must face that possibility.

In view of the unusual nature of this case, both clinically and pathologically, we have made great efforts to get a detailed history. I have looked up the mother of the child and questioned her carefully. For a long time she was unco-operative, but she finally confessed that the injections were given not by a physician, but by her brother, a pharmacist. He works in some laboratory out of town, but she was not clear as to which laboratory it was. In view of this additional history it becomes a possibility that this pharmacist might have made an error and taken the wrong mixture from his laboratory, taking diphtheria toxin instead of T and A mixture. Such a possibility would be consistent with may of the pathologic findings, namely, the myo-

carditis and nephritis.   That would not explain the cerebral
hemorrhage as far as I know.   The thought occurs to me that
perhaps with injured blood-vessels the blood transfusion might
have been responsible.

There is also the possibility that the antitoxin in the T and A
mixture had become inactive, leaving the toxin free, with the
result that the child received pure diphtheria toxin.

At any rate, although the answer is not quite clear, I feel
justified in submitting this case to your consideration for the
following reasons:

1. If it was due to the T and A mixture, it certainly should
go on record, so that other men may watch for similar occur-
rences.

2. If it was due to the T and A mixture's being given to a
child with nephritis, it certainly emphasizes that great caution
should be employed in using this mixture under such circum-
stances.

3. It suggests the serious results that may follow attempts
at medication by untrained individuals.

4. It emphasizes the importance of a very careful history.
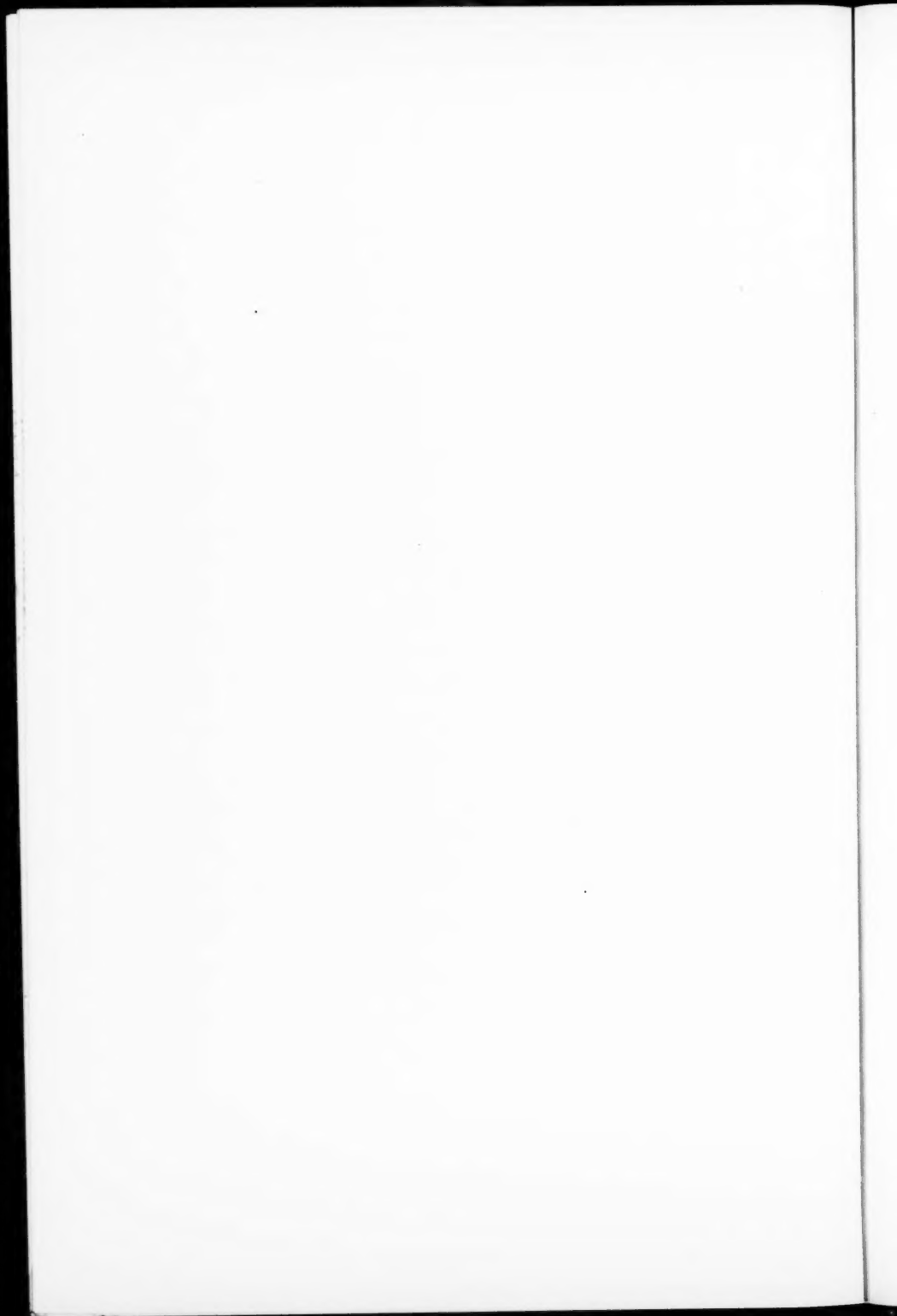
CLINIC OF DR. GEORGE O. SOLEM

WASHINGTON BOULEVARD HOSPITAL

DUODENAL OBSTRUCTION DUE TO CHOLECYSTODUO-DENOCHOLIC BAND

M. L. HARRIS in 1914 reported 6 cases with indefinite distress in the upper abdomen, where the only pathology found was a band extending from the gall-bladder and under surface of the liver to the duodenum and transverse colon. These bands apparently caused a constriction of the duodenum in its second and third portions. In most of these the diagnosis was made before operation. Relief of symptoms was obtained after severing this band. He identified this band as one of the accessory ligaments of the liver, and described and illustrated its embryology, tracing it to abnormal remains of the caudal edge of the ventral mesagastrium.

Since then a number of men, particularly in the East, have reported similar cases, but I feel that this condition has not received the attention which it deserves in the differential diagnosis of upper abdominal distress. Poirer and Charpy, in their Anatomy, published in 1901, describe the hepatoduodenal ligament, the suspensor of the duodenum, as the free edge of the gastroheptaic omentum, and also describe its prolongation to the right, in a certain percentage of cases, as a ligament which extends from the gall-bladder above to the descending portions of the duodenum and the hepatic angle of the colon, and call this prolongation the cysticoduodenal and cysticocolic ligaments.

Ancel and Sencert show that it develops together with the greater and lesser omentum, and call it the cysticoduodeno-omental ligament. They report its occurrence in 29 per cent. of cases. They found it in the fetus as early as the seventh month, but it is rarely fully developed until early childhood. They con-

sider it as complete when it extends to within 1 cm. of the fundus of the gall-bladder, and state that it may extend beyond the fundus on to the free edge of the liver, as it did in one of my cases when it caused the gall-bladder to be definitely lobulated.

Nagel, at the Mayo Clinic, recently reported its occurrence in 12 per cent. of 150 autopsies, and various other authors have reported its occurrence in 20 to 30 per cent. of cases.

Bryant in 1922 found that in 34 autopsies on the fetus only 5.9 per cent. were free from demonstrable adhesions or bands, but these were less complex than those found later in life. He found the band extending from the gall-bladder to the duodenum and colon the most frequently.

In 1905 Robert A. Morris wrote a paper entitled "Gall Spiders," in which he described adhesions present in the upper abdomen, which he referred to as cobwebs in the attic of the abdomen, and suggested that these might explain the cases where good results were obtained by operation where pathology adequate to explain the symptoms was not found, but where the patient had complete relief. He considered these bands as inflammatory and secondary to low-grade infections in the gall-bladder or about the duodenum.

Homans in 1916 reported 11 cases where this band was found at operation, but not causing definite constriction, although several of his x-ray pictures showed definite dilatation of the first and second portions of the duodenum. All were relieved after the band was cut, but in all some other operative procedure was carried out, so that the results cannot be accepted as due wholly to severing the band.

Lewis Cole in 1922 called attention to Harris bands, but presented a number of sketches and x-ray pictures, showing that the veils were not as uniform as described by Harris. He stated that they often cause deformity of the antrum and involve the first rather than the second and third portions of the duodenum.

Hamann in 1922 reported 3 cases where operation was done for gall-bladder or ulcer, and nothing found but this band, but relief was obtained by its severance. Cromarty in 1922 reported a number of cases of indefinite abdominal distress where, on

fluoroscopic examination, were seen deformities of the first and second portions of the duodenum, which were interpreted as bands and where relief was obtained after operation. Walter Niles in 1924 reported 4 cases operated, with a diagnosis of a band causing partial obstruction with relief after operation. In these cases there was fixation of the duodenum in the first and second portions. Alfred Taylor reported rather a large series of cases with symptoms due to anomalous membranes where he described the Harris bands, the pericolic membrane, and the duodenojejunal membrane. I wish to report 3 cases where definite obstruction of the duodenum was caused by these bands.

Miss C. P., age twenty-three, was referred to me by Dr. Paul Fox because of an attack with persistent vomiting and pain in abdomen.

Vomiting began suddenly five years ago, continued for four or five days, when she was operated for appendicitis, but the patient continued vomiting until she left the hospital, when she gradually improved, so that she vomited only once a day, usually after breakfast, but continued to have distress and vomiting at intervals. At times she vomited food eaten the day before.

Three years ago, after another severe attack of vomiting, she went to a hospital for examination of the kidneys, but was discharged after four or five days and told that the kidneys were normal. Four months ago she was taken to another hospital and operated for adhesions about cecum, but with no improvement. Another attack of vomiting occurred about one month ago. Patient was taken to a hospital and x-rayed, and told she had prolapse of the stomach, and was advised to have a gastropexy. Pain in abdomen had been increasing in severity with each attack. It was described as a sticking pain which came on just before vomiting, and was gradually relieved after vomiting. Pain was not definitely localized, but her impression was that it was most severe in epigastrium. It usually came on soon after eating. She had lost 15 pounds in the past four months and 32 pounds since the onset five years ago.

Past and family history were negative.

Examination revealed a young woman in bed, apparently

ill. Patient was undernourished and dehydrated, with dry tongue, sunken eyes, cracked skin, and fissured lips. Patient was moaning and retching at times. Chest was negative. Pulse somewhat rapid. Reflexes were normal. Abdomen presented a right rectus and midline scar below the level of the umbilicus. There were no herniæ. No masses were felt. Colon was somewhat tender along its entire course.

Peristaltic waves of high grade were seen passing from left to right about the level of the umbilicus, following the course of stomach hyperperistalsis. During the examination the patient vomited about a pint of greenish-yellow material having the odor of small intestine contents. On examination the epithelial cells were seen to be bile stained. There was a definite chemical reaction for bile with the nitric acid test. There was no free acid in the vomited material. No small intestine bacteria were seen. Before I saw her an Ewald test-breakfast had been given, and aspiration at the end of an hour obtained 200 c.c. of material, 25 per cent. food. Free HCl 28, total 50. No blood. Red blood-count 4,320,000, white blood-count 7500, and hemoglobin 80. Urine was negative. Stools showed no blood, pus, or mucus. A tentative diagnosis of partial obstruction of the lower duodenum or upper jejunum was made. The patient was given proctocylsis to overcome the dehydration and the next morning was fluoroscoped.

The duodenal cap was apparently normal in size and smooth in outline, but as the cap emptied the material was retained in what was apparently the first 3 or 4 inches of the duodenum. Vigorous peristalsis and antiperistalsis were seen in this portion, but by pressing upward on this area the material passed very quickly beyond the point of apparent obstruction at about the middle of the third portion. After seven hours the stomach still retained about one-third of the barium. At several observations during this time the duodenum was seen to be about half-filled with barium in its proximal portion.

At operation there was found a band extending from the gall-bladder to the second and third portions of the duodenum and down to the colon. This band was cut and abdomen closed.

Convalescence was rapid and uneventful. She had usual gas pains for several days, but no vomiting. She was last seen over a year after the operation, had gained 35 pounds, and had had no recurrence of vomiting or distress.

Mr. H. E., age thirty-nine, came complaining of abdominal distress, described as a feeling of fulness and distention, as though there was a weight in the abdomen. At times the distress would be cramp-like and shifting from left to right at level of umbilicus. The distress was worse after a large meal, but was present all day. This distress had been persistent for twenty years and he had had to limit his work, as he noticed that he was more likely to have distress after doing heavy work. He had learned to restrict his diet because of the greater distress after large meals. Distress was aggravated by lying down. Distress often awakened him at 10.30 P. M., when he would frequently vomit food eaten at 6.30 P. M. Vomiting would give complete relief. Taking of food when distress was present would increase the distress. His bowels were regular and there was no history of laxative taking.

Examination was essentially negative except for slight tenderness of descending colon, which was somewhat spastic. On distending the stomach with sodium bicarbonate and tartaric acid, definite peristaltic waves were seen passing from left to right. There were no other findings of any significance. An Ewald test-breakfast aspirated at the end of an hour contained 225 c.c. of material with 80 per cent. food. Free HCl 18, total 32. The stomach was empty seven hours after a large meal. At the time patient was having distress a therapeutic aspiration was done and the patient obtained relief. Titration of the material aspirated showed a free acidity of only 38 in terms of decinormal NaOH. While under observation it was noted that while the visible peristaltic waves were of a height not seen except with obstruction, the distress was not compatible with ulcer, in that it came on too soon after eating, was made worse by taking food, and was increased after lying down. There was no blood found in stomach contents or in several stools which would speak against malignancy of the gastro-intestinal tract. There was no

history suggestive of gall-bladder disease. The patient was fluoroscoped to determine the point of obstruction.

The stomach was vertical, J shaped, with the lower border at the level of the symphysis. It filled without evidence of defect and there was no retention of opaque material along its walls. Peristaltic waves of considerable depth were seen to close off well at the outlet. The duodenal cap filled well. Upon emptying, a fleck of opaque material was seen to remain which could not be displaced by palpation. The patient experienced some tenderness over this point, which would be at the base of the cap. The opaque material was seen to be checked at a point 6 cm. from the pylorus. In this portion distended by barium there was vigorous peristalsis and antiperistalsis, forcing part of the material back into the stomach at times. Check up at the end of four hours showed the stomach about half-empty and the dilated portion of the duodenum was partly filled with barium. At the end of seven hours the stomach was completely empty, but there was still some material in the duodenum.

A diagnosis of partial obstruction of duodenum due to cystico-duodenocolic band was made and operation advised. · Operation was done by Drs. Lounsbury and Metz. An upper midline incision was made. The antrum and duodenum were examined first from above, and then the omentum and transverse colon were raised and explored with the fingers. Some of the bands anchoring the duodenum at this point were cut. The omentum and transverse colon were then placed back in the abdomen and the gall-bladder was examined. A band was found extending across the body and fundus of the gall-bladder to free edge of the liver, tending to lobulate the gall-bladder, and down to the duodenum. These were severed. On palpation the gall-bladder wall and ducts seemed normal.

The patient made an uneventful recovery and had no more of this distress after his operation. He returned six months later because of a bowel distress in left lower quadrant, but had been entirely free from the distress that had been present for twenty years.

Mr. A. S., age twenty-seven, entered the Washington Boule-

vard Hospital on Dr. B. W. Sippy's service. About two years ago he began to be awakened at 4 or 5 A. M. with epigastric distress. At this time he went to a physician, who x-rayed him and gave him test-meals, and told him he had hyperacidity and probably gastric ulcer, and put him on a diet of milk and cream and cereal. He had relief for a period of four months, but the night distress has been present from time to time since. This distress he described as a feeling of fulness and pressure. The distress was definitely localized to epigastrium, and when present during the day would come on almost immediately after meals and last two to three hours. Passage of flatus would not relieve. There was no rumbling or gurgling associated with this distress.

He also had another type of distress in lower abdomen, which . was definitely different and due to taking cathartics. He had been on a rather limited diet and had lost 20 pounds before entering the hospital. Past and family history were essentially negative.

Physical Examination.—A fairly well-developed male of Stiller type, with some evidence of recent loss of weight, apparently not acutely ill. Heart and lungs were negative. Tonsils were slightly enlarged. Reflexes normal. There were no masses or tenderness in abdomen, but at the time of the first examination about two hours after a full meal there was seen definite peristaltic waves passing from left to right of such height as to speak for a definite obstruction. An Ewald test-breakfast removed at the end of an hour contained 25 c.c. of material with 70 per cent. food. Free HCl 22, total 62. There was a small amount of residue obtained seven hours after a large meal.

Under observation it was proved that this distress was incompatible with ulcer and patient was fluoroscoped to determine the point of obstruction. x-Ray findings were as follows:

The stomach was vertical, J shaped, with the lower border 4 cm. below the umbilicus. It filled without evidence of defect and there was no retention of opaque material along its walls. Very good peristaltic waves closed off sharply at the outlet. The duodenal cap filled well and appeared normal in outline, size, and position. The opaque material was observed to pass

on into the first and second portion of the duodenum and then upward into the first two or three cm. of the third portion, beyond which point the opaque material was not observed to pass. Reversed peristalsis was observed to pass backward from this point toward the duodenal cap. Pressure over this apparent point of obstruction caused the patient slight discomfort. Checkup examination at the end of five hours showed the stomach two-thirds empty and the opaque material well along in the small bowel. There was stasis in the portion of the duodenum proximal to the obstruction as observed in the other 2 cases.

A diagnosis of partial obstruction of duodenum due to cystico-duodenocolic band was made, and operation advised.

Operation was performed by Drs. Lounsbury and Metz. The abdomen was opened by a midline incision extending from near the xyphoid process to the umbilicus. A semitranslucent band was found extending from the fundus and body of the gall-bladder to the omentum and transverse colon. After the duodenum had been exposed it was seen that this same band was continued over the duodenum in this region. The gall-bladder and pylorus were normal. These several bands or the many parts of the same band were all severed, and the peritoneum, fascia, and skin were all closed in the usual manner. The patient made an uneventful recovery and has had no recurrence of his distress.

The symptoms and physical findings in these cases showed a certain similarity which is worth noting. The distress was in upper abdomen, but not definitely localized. It came on shortly after eating and continued for two to three hours. This would correspond to the time when stomach peristalsis would be most active and the tension in the duodenum would be greatest.

This time relation to meals was noted by Alfred Taylor in 1922 in a larger series of cases of this type.

The greatest relief is obtained by vomiting, thus emptying the stomach. Vomiting may or may not be associated with nausea. Eating at time of distress increases it. Small, frequent meals gave complete relief for a time in 2 of my cases. In 1 of my cases the distress was worse on lying down; 2 had distress

at night. The distress usually begins in the second or third decade of life, but other men have reported its occurrence in all ages. The attacks of distress had a tendency to be periodic. This was brought out by Holland in a recent paper. The patient is more likely to have distress when overworked or tired. Loss of weight also seems to predispose to an attack. The history is essentially that of a chronic disorder, with a definite tendency to become worse.

Gibson states that hypo-acidity is practically a constant feature. In 12 cases of his series, where gastric analyses were made, only 1 had hyperacidity and 5 had anacidity. In my cases the Ewald results were as follows:

C. P.	200 c.c. material	HCl 28–50
H. E.	225 c.c. material	HCl 18–32
A. S.	25 c.c. material	HCl 22–62

In the third case the stomach was probably not completely emptied, as the material contained 70 per cent. food, and there was some residue in the stomach seven hours after a large meal.

The quantity obtained would suggest delayed emptying, as the usual Ewald removed at the end of an hour will contain from 75 to 150 c.c. of material.

An important diagnostic point, which has probably not been stressed, was the presence of visible peristalsis in all 3 cases. In only 1 case was sodium bicarbonate and tartaric acid given to distend the stomach before the waves were seen. These waves were of such height as to be definite evidence of obstruction, as the abdominal wall was of normal thickness. Visible peristaltic waves may be seen with normal stomach musculature when there is a marked diastasis recti or a very marked thinning of the abdominal wall.

I do not feel that bands such as described should be assumed to be causing distress unless there can be demonstrated definite obstruction, as evidenced by visible peristaltic waves.

The x-ray findings in these 3 cases were very similar, in that the duodenal cap filled well and the duodenum was dilated in its first and second portions. Vigorous peristalsis and reverse

peristalsis were seen in the dilated portion. Later the barium was seen to pass the point of obstruction into the small intestine, but the stomach was not completely empty at the end of five hours. In only 1 of the 2 cases where their emptying power was tested with a large meal was there any residue after seven hours.

The question arises as to whether the symptoms in cases of this type are due to the obstruction or to traction on the liver or gall-bladder. No evidence of gall-bladder disease was found in these cases. I would be inclined to believe that the symptoms are due to obstruction because I can recall having seen cases where the duodenum was held close to the gall-bladder, and greater traction would be expected, but with no symptoms. In my cases the greatest distress occurred in the case with the highest grade of obstruction.

Holland states that in intestinal motility the greatest tension is maintained in the duodenum, and suggests that the distress might be due to the limitation of expansion of the duodenum.

It is impossible to draw any definite conclusions from the relatively few cases of this type reported, but there are certain features which are fairly constant and should draw our attention to the possibility of this band.

1. The periodic recurrence of attacks of epigastric distress coming on almost immediatley after eating and continuing until the stomach is practically empty.

2. The presence of visible peristaltic waves.

3. The characteristic x-ray findings of obstruction in the second or third portions of the duodenum.

4. The finding of normal acidity or hypo-acidity in the presence of obstruction.

CLINIC OF DR. ISADORE PILOT

Michael Reese Hospital

FUSOSPIROCHETAL PULMONARY INFECTION COMPLICATING DIABETES MELLITUS

PULMONARY complications in diabetic patients develop commonly, and in spite of insulin therapy prove very serious. The formation of an abscess or of a gangrene lesion is particularly dreaded. In a previous study[1] it was pointed out that in pulmonary abscess and gangrene certain anaërobes were prevalent in the tissues. These organisms are classified in the group included under Bacillus fusiformis and the symbiotic Spirochæta vincentini. For the development of pulmonary infection with these bacteria many predisposing factors, such as general anesthesia, tonsillectomy, bronchiectasis, pulmonary carcinoma, are often the important determining factor. In one patient diabetes mellitus appeared to be the outstanding contributing cause. Subsequently 2 other diabetic patients were observed with pulmonary gangrene in which these anaërobes were the predominant bacterial agents.

Case I.—Ref. I, age fifty, colored, male, entered the Cook County Hospital complaining of pain in the right chest, cough, expectoration of foul sputum for one month. He had known he was a diabetic for two years, with symptoms of thirst, polyuria, and loss of weight. Upon examination the patient was emaciated, dyspneic, and drowsy; the teeth were false, breath very foul. Dulness, increased fremitus, moist râles, with friction-rub were found over right lung. The urine contained large amounts of glucose, acetone, diacetic acid, and trace of albumin.

[1] Studies of Fusiform Bacilli and Spirochetes: IX. Their Rôle in Pulmonary Abscess, Gangrene, and Bronchiectasis, Pilot, I., and Davis, D. J., Archives of Internal Medicine, 1924, 34, pp. 313–354.

The patient lapsed into coma the following day, although insulin and glucose were administered, and died within twenty-four hours. At autopsy there was a confluent bronchopneumonia of both upper and lower lobes of right lung, with multiple foci of gangrene, lobar consolidation of middle lobe with a gangrenous abscess, diffuse putrid bronchitis, acute serofibrinous pericarditis, atrophy of pancreas. The pleura over the right lower lobe was covered with fibrinous exudate. The gangrenous areas measured 1 to 3 cm. in diameter, were of green color, and filled with very foul exudate. Smears from these areas revealed many fusiform bacilli, cocci, and few spirochetes. In Levaditi preparations the bacilli and cocci predominated in the necrotic masses, but at the margin they became less common and the spirochetes very numerous, migrating into the surrounding tissues. In cultures fusiform bacilli, Streptococcus hemolyticus, a few colonies of Staphylococcus albus, and pneumococcus were identified. From these observations it was evident that the diabetes predisposed the patient to a rapidly progressive form of fusospirochetal pulmonary gangrene.

Case II.—S. R., aged fifty-eight, white male, entered the Michael Reese Hospital with complaints of a "diabetes" of ten years' duration, recent symptoms of pain in chest, and cough for two weeks. Drowsiness was noted for three days. About thirty years ago he had developed a lung infection (abscess?), with symptoms of cough, hemoptysis, and purulent expectoration. He apparently improved, although for many years he complained of persistent cough. Ten years ago he developed mild symptoms of diabetes which was fairly well controlled by restricted diet. Except for occasional glycosuria he was in good condition until he was exposed two weeks ago to "cold draft," and developed on the following day pain in right chest of a severe stabbing character. Cough became marked, and pain continued until three days before entrance, when he noted less pain, but more dyspnea, decidedly foul expectoration, and drowsiness. The significant findings were: temperature 101° F., pulse 88, respiration 44. The right chest was flat over lower

lobe, together with diminished fremitus and breath sounds. The left chest was hyperresonant. The fingers were cyanotic and moderately clubbed. The peripheral blood-vessels are sclerotic. The sputum was foul, profuse, grayish green, and in smears revealed numerous fusiform bacilli, small Gram-negative bacilli and Gram-positive cocci, but no tubercle bacilli. The blood examination presented 80 per cent. hemoglobin, 4,445,000 red blood-cells, and 15,000 leukocytes, of which 70 per cent. were polynuclear neutrophils; blood-sugar was 361 mg.; non-protein nitrogen 53 mg. On diet of caloric value varying from 700 to 1000 calories he excreted 40 to 60 grams of glucose, acetone, and diacetic acid in spite of 60 units of insulin daily. The insulin was increased to 90 and 120 units and the drowsiness diminished, diacetic acid disappeared, but glucose and acetone persisted. The expectoration became more profuse and foul, and the flatness rose to the level of sixth dorsal vertebra posteriorly. Paracentesis was performed and foul pus obtained. A closed method of drainage was instituted by Dr. Bettman because of the desperate condition, and from 3 to 4 ounces of pus aspirated every two hours, followed by irrigation with Dakin's solution. In the pus from smears and cultures Bacillus fusiformis, B. coli, and Streptococcus viridans were identified. Glycosuria persisted in spite of 140 units of insulin; the patient became more toxic and died six days after admittance.

At autopsy the right pleural cavity was filled with 600 c.c. of thick, green, foul pus. The right lung was green; the bronchi of lung were large and in the lower lobe was an old abscess cavity encapsulated by fibrous tissue containing a broken molar tooth. The anatomic findings were summarized: gangrene and purulent empyema of the right lung; perforation of the pleura with skin emphysema; compression atelectasis of right lung; chronic interstitial pneumonia of right lower lobe; purulent confluent bronchitis, peribronchitis and bronchopneumonia of the middle lobe; foreign body (tooth) in lower part of lower lobe in old abscess cavity; bronchitis and bronchopneumonia of left lower lobe; parenchymatous degeneration of left kidney with purulent pyelitis; amyloid spleen; nutmeg liver.

Cultures of pus from pleural cavity revealed fusiform bacilli, Bacillus coli, and streptococci. In Levaditi preparations of the gangrenous portion of lung fusiform bacilli and cocci occurred, but no spirochetes were found.

In this patient early in his life he had apparently aspirated a tooth, resulting in the formation of a chronic lung abscess. The abscess cavity became well encapsulated, but remained as a smoldering infected area. When the resistance became sufficiently lowered by the diabetic state the micro-organisms rapidly spread into the lung, causing gangrene and perforation of the pleura with the development of a putrid empyema.

Case III.—C. M., aged twenty-five, white male, entered the Lutheran Memorial Hospital complaining of pain in left chest for two days. He stated he had chills four days previously and some soreness in the throat. The pain was of a sharp, stabbing character, worse on coughing. In his past history it was ascertained that he had a moderately severe diabetes for four years, which was being controlled in the past year by insulin. He admitted that he was negligent in his diet and that he frequently passed large quantities of glucose in the urine. Upon examination patient was very emaciated (85 pounds). Temperature 103° F., pulse 130, respirations 30. Throat was red, tonsils were absent; teeth were in fair condition. Distinct signs of consolidation appeared over left lower lobe, and on the third day a diagnosis of lobar pneumonia was made. The sputum was at first tinged with fresh blood and later became rusty. Urine contained 3.3 per cent. glucose, acetone, no diacetic acid; hemoglobin was 80 per cent, red cells 4,650,000, leukocytes 12,100, of which 86 per cent. were neutrophils. Throat smears and culture contained no diphtheria bacilli or Vincent's organisms. The sputum revealed streptococci and pneumococci, but no tubercle bacilli. He was placed on a balanced diet of 1000 calories and received 20 units of insulin daily. The urine contained from 1 to 5 gm. of glucose daily and no acetone. The temperature continued high for six days (103° to 104° F.), pulse 120 to 130, and râles appeared in the left chest. On the

ninth day the sputum became distinctly foul and liquid green. Smears revealed many fusiform bacilli, few spirochetes, many streptococci, and no tubercle bacilli. *x*-Ray presented an unresolved pneumonic process with abscess formation and cavitation, one 4 by 6 cm. and one 1 cm. in diameter in the left lower lobe. He was given 5 grains of sodium cacodylate daily, and the sputum became less foul, but still copious. Insulin was continued daily at 20 units. The urine became sugar free; bloodsugar 73 mg. He continued, however, without further improvement, and died on the sixteenth day after admission. Autopsy could not be obtained.

In this patient the diabetes was the underlying predisposing factor of the complication of gangrene following lobar pneumonia. The gangrene developed, although the diabetes was apparently under control with insulin.

Discussion.—The sources of infection in fusospirochetal pulmonary gangrene are largely the bacteria that reside about the teeth, tonsils, and nasopharynx. In previous investigations it was pointed out that the tonsillar crypts as well as the tartar of the teeth often harbor large numbers of fusiform bacilli, spirochetes, and streptococci. These organisms apparently extend into the lower respiratory tract without the development of typical Vincent's angina, and give rise to a necrotic lesion if the bronchi and parenchyma are susceptible. In one instance the lobar pneumonia and in another a foreign body (tooth) diminished the resistance of the pulmonary tissue. These illustrate that multiple factors often underly the complication of abscess and gangrene due to the anaërobes.

The symptoms are gradual at the onset, with fever and pain in chest. Expectoration does not become foul until a lapse of eight to fifteen days. The course in the diabetic is rapid. The lesions usually appear in the form of multiple foci of gangrene, with early cavity formation. There is no tendency toward localized abscess formation, but instead often rapid extension to the pleura, perforation, and the development of a putrid empyema.

The diagnosis, as a rule, is not apparent until the patient be-

gins to expectorate foul sputum. The breath is foul and the sputum profuse, green, intensely fetid. Elastic tissue and necrotic lung parenchyma may be found. In smears stained with 10 per cent. carbolfuchsin or by the Fontana method there are enormous numbers of bacteria, particularly fusiform bacilli, spirochetes, together with pyogenic micro-organism, such as streptococci, pneumococci, and staphylococci. The spirochetes are morphologically like those observed in Vincent's angina and entirely unlike the spirochetes of syphilis.

It is evident that, if the mouth is the important source of these organisms, the proper and thorough hygiene of the teeth and throat is especially indicated in the diabetic. Tartar deposits and carious teeth harbor large numbers of these bacteria. The tonsils are often filled with firm plugs that may be expressed from the crypts. Frequent cleansing of the mouth in a diabetic patient who develops a pulmonary infection or is to be given an anesthetic may diminish the danger of complications of abscess or gangrene.

In the treatment of fusospirochetal pulmonary infections neosalvarsan or other arsenicals have been employed with beneficial results in some cases. In one of the diabetic patients (Case III) the sputum became less offensive after sodium cacodylate injections, but the lesion continued to progress. It is doubtful if salvarsan even when combined with insulin therapy will prevent a fatal outcome in a diabetic with pulmonary gangrene. Often the insulin in large doses will not prevent acidosis or influence the rapid extension of a pneumonic process. If the diagnosis is made early, the combined use of these remedies should be employed in the hope of arresting the process before the lesions become far advanced and beyond surgical measures.

CLINIC OF DR. N. C. GILBERT

ANGINA PECTORIS

I WISH to give this clinic without showing a case, for one of the points I wish to emphasize is that there is nothing to be seen on inspection, or to be found on careful physical examination, by means of which a diagnosis of angina pectoris may even be inferred, unless the patient be actually seen during an attack. What you learn from the patient must be from the patient's own story, and his own words will seldom leave you in doubt. But where there are so many variations, so many elisions, I would not wish to read a history as typical. I would prefer, in fact, to quote to you, as many have before me, part of Heberden's account. It is about a perfect description of the disease, to which, after one hundred and fifty-seven years, we can add but little. It will help us to start with a clear conception of just what we are to discuss.

"But there is a disorder of the breast marked with strong and peculiar symptoms considerable for the kind of danger belonging to it, and not extremely rare, which deserves to be mentioned here at length. The seat of it, and sense of strangling and anxiety with which it is attended, may make it not improperly be called angina pectoris.

"They who are afflicted with it are seized while they are walking (more especially if it be up hill and soon after eating) with a painful and most disagreeable sensation in the breast, which seems as if it would extinguish life if it were to increase or to continue; but the moment they stand still all this uneasiness vanishes.

"In all other respects the patients are, at the beginning of the disorder, perfectly well, and, in particular, have no shortness of

breath, from which it is totally different. The pain is sometimes situated in the upper part, sometimes in the middle, sometimes at the bottom of the os sterni, and often more inclined to the left than to the right side. It likewise very frequently extends from the breast to the middle of the arm. The pulse is, at least sometimes, not disturbed by this pain, as I have had opportunities of observing by feeling the pulse during the paroxysm. Males are most liable to this disease, especially such as have passed their fiftieth year. After it has continued a year or more, it will not cease as instantaneously upon standing still, and it will come on not only when the persons are walking, but when they are lying down, especially if they lie on the left side, and oblige them to rise out of their beds. In some inveterate cases it has been brought on by the motion of a horse or a carriage, and even by swallowing, coughing, going to stool, speaking, or any disturbance of mind.

"Such is the usual appearance of this disease, but some varieties may be met with. Some have been seized while they were standing still or sitting, also upon first waking out of sleep, and the pain sometimes reaches down the right arm as well as the left, and even down to the hands, but this is uncommon; in a very few persons the arm has at the same time been numbed and swelled. In one or two persons the pain has lasted some hours or even days, but this has happened when the complaint has been of long standing and thoroughly rooted in the constitution; once only the very first attack continued the whole night.

"I have seen nearly a hundred people under this disorder, of which number there have been 3 women and 1 boy two years old. All the rest were men near or past the fiftieth year of age.

"Persons who have persevered in walking till the pain has returned four or five times have then sometimes vomited. The termination of angina pectoris is remarkable. For, if no accident intervene, but the disease go on to its height, the patients all suddenly fall down and perish almost immediately. The angina pectoris, as far as I have been able to investigate, belongs to the class of spasmotics, not of inflammatory complaints."

What I have just quoted is taken from Heberden's "Com-

mentaries," written in 1782, differing little from his original address.

Accounts of what were probably attacks of angina pectoris had appeared at intervals from the time of Hippocrates. Morgagni in 1707 described what was undoubtedly such an attack. Earlier in 1768 Rougnon, in France, had described an attack in a letter to a friend, which was published. But it was Heberden who first clearly differentiated it as a diagnostic entity, and who gave it its name. I wish that time permitted us to go into the history of angina, for I know of no disease which has left us descriptions so well written and concerning men so well known to you in the history of medicine, literature, and statesmanship. You will find references to the earlier history of angina in Sir M. Gairdner's section on angina in Reynold's System, in the addresses of Gibson, and in Osler's lectures on the subject. And if you follow still further the clues to the literature which you thus find your time will be well rewarded.

Whether angina pectoris should or should not be regarded as a disease one cannot discuss here further than to say that the question has been, and still is, considered a matter for discussion. We can at least agree that it is a diagnostic entity. Says Latham, "We are sure of what it is as a collection of symptoms—we are not sure of what it is as a disease."

If we were to be asked to select from the symptoms given by Heberden that which we considered the most characteristic, I think that we would agree that it is not the pain itself, for somewhat similar pain may arise under different conditions, and more severe pain in many. What is more characteristic is the "sense of strangling and anxiety," what has been referred to as the "essential unbearableness" of the pain, something more than the pain itself, but a sense of dread, something, as Latham says, "which is beyond the nature of pain, a sense of dying." Seneca, in his fifty-fourth letter to Lucillius, writing of his illness, says, "Its attack is of very brief duration, like that of a squall at sea; it usually ends within an hour. Who, indeed, could breathe his last for long? I have undergone all bodily ills and infirmities, but none appears more grievous. Why not? Because to have

any other malady is only to be sick, to have this is to be dying.
Hence physicians call it 'practising in death.'"

With this anxiety, or dread, is a sense of oppression, of con-
striction, of not being able to breathe. "As though my sternum
were being drawn back to my spine," John Hunter said. Mac-
kenzie inclines to the view that there is an actual contraction of
the intercostal muscles. Hunter, observing himself in a glass
during an attack, noticed that he was not breathing. Certainly
in some there is an apnea, or at least a diminished respiratory
rate. Others observed during an attack are seen to breathe nor-
mally, yet complain of the same sense of compression, of not
being able to breathe. It is a symptom which one must guard
against misinterpreting in listening to the patient's story of an
attack, as the patient's statement that he was "breathless"
may be taken to mean that he was dyspneic, unless the patient
be further questioned.

The sense of dread and the sense of oppression or constric-
tion have no relation to the severity of the pain. The pain may
be slight, or one or the other, but more frequently both together,
may appear without any pain, and may be the only symptom
in a fatal attack. As we may have the sense of oppression and
the sense of dread without the pain, so we may have the pain
alone, although most typically they are associated.

The pain itself is typically substernal, and most often under
the upper third, or just to the left of the upper third of the
sternum, but it may appear lower down under the sternum, or
at the xyphoid, or it may appear only in its peripheral radiation
and not under the sternum at all. Precordial pain does occur,
but is not characteristic, and, as Heatherby has pointed out, is
more frequent in cases which are purely on a neurogenic basis.

The pain may be intense, agonizing, or a dull ache, or of a
burning character, and may be very slight, even in fatal attacks.
It is never a short, stabbing pain.

The pain may remain substernal or it may radiate to the left
side, to the left neck or shoulder, or down the left arm, but it
may radiate to the right side or to both, and frequently to the
epigastrium or right costal border. In the arm it is more apt to

follow the ulnar distribution. It may radiate all the way down to the fingers, or only part way, or to the shoulder, elbow, or wrist. The pain may be localized in one area, as in the little finger, or wrist, or elbow, without any substernal pain, or any radiation. A patient may state that on effort, as climbing stairs, he has pain in his little finger, or feels a tight band about the wrist, and that, after the pain goes, the part feels swollen or numb, although it is quite normal. Occasionally the pain may appear only in the thumb, or it may be one condyle of the elbow, or some other area on the arm. One case had a sense of mild warmth over the outer condyle if he walked too rapidly, which would disappear if he stopped or walked more slowly, but which would become such an intense, burning pain if he continued that it would force him to stop.

Pain referred to the lower angle of the jaw, to the mastoid region, along the sternocleidomastoid muscle, or to the lower left molars is not uncommon. Radiation to the upper abdomen, to the epigastrium or along the right costal border is frequent. Such pain has been mistaken for gall-stones as well as accounting for at least many of the deaths due to "acute gastritis."

Cases are reported of more unusual radiation, as to the lumbar region or the testicles. Von Neusser mentions it being mistaken for kidney colic.

Pallor is frequent during the attack and with it a profuse, cold perspiration. Then, as the attack subsides, the color returns and the face flushes. Or there may be a redness of the hands and arms, and a flush of the face, extending over the chest, described by Nothnagel as the "vasomotor type."

Dysphagia may be present in angina patients, occurring with the attacks, or preceding the attacks, or may be present without reference to an attack. Usually, when dysphagia is present, eating will bring on an attack. During the periods when dysphagia is present attacks are usually more frequent.

Other vagal symptoms, as aphonia or hoarseness, are reported.

Belching of gas is frequent, especially as the attack ends. The patient may be sitting up, in obvious pain and anxiety, the

face pallid and the skin perspiring, when he may belch a quantity of gas, the face flushes, an appearance of great relief appears, and the patient announces that the attack is over.

There is nothing characteristic about the pulse. The blood-pressure may be high, low, or normal, or during the attack it may be higher or lower than the normal for the individual, or it may be unchanged. The rhythm is normal and the electrocardiogram reveals nothing characteristic.

Respiration is usually unaffected. If affected at all, it is more apt to be slowed. Dyspnea is always a complication as far as the attack itself goes, although cases are frequent enough when we have cardiac dyspnea and angina in the same patient. As Von Neusser points out, the angina frequently disappears as the evidences of broken compensation appear, to reappear as the heart improves under rest and treatment.

Attacks are most frequently consequent upon exertion, and especially exertion after meals. A patient who cannot walk but a short distance without bringing on an attack after a meal may walk miles on an empty stomach with no discomfort. There may be no pain when walking on the level, or the pain appear on walking up a slight incline, or stairs, or walking facing a wind. A patient may have pain when he is making a conscious effort to keep up with a companion, even though he be walking at a gait which would not otherwise elicit pain, or he may have pain walking and be able to ride a bicycle in comfort.

The French authors speak of an angina of effort and an angina of decubitus. Intense anginal pain, coming on at night and arousing the patient from sleep, is a frequent enough observation, especially in those with low blood-pressure. The attacks may come only at night from the first, or, as Heberden says, a patient who at first has attacks only on effort during the day may later develop nocturnal attacks. Vaquez considers that he has shown dilatation of the heart in such cases by orthodiascopy. We know that the coronary flow is a direct function of blood-pressure, and it is much easier for one at least to assume that the nocturnal pain is due to a decreased coronary circulation,

due to a fall in blood-pressure in the presence of sclerosed coronaries.

Emotion very frequently brings on an attack, especially anger, worry, anxiety, grief, or mental excitement of any origin. A case seen recently came on after severe business losses, another after the sudden death of an only son. Anger frequently enough precipitates an attack. John Hunter said that his "life was in the hands of any rascal who chose to annoy and tease him," and, as it turned out, he spoke truly.

Flatulence also seems to be an exciting cause, and an amount of exertion which would ordinarily not bring on an attack may do so in the presence of flatulence.

In some patients the pain is most frequent in the forenoon, especially after breakfast. In others it is most frequent at night, especially when fatigued or worried, or after a full evening meal. Attacks may be brought on by defecation or by urination, especially when arising in the morning and emptying a full bladder.

Many patients are less disposed to have attacks in warm weather. In a smaller number a hot day may bring on more frequent attacks.

So much for the typical attacks. False angina or functional angina we should be slow to diagnose. Many and many a case of false angina has died just as dead during an attack as though it had been diagnosed as of the orthodox faith. It is well to remember Potain's dictum, that there are false diagnoses, but not false diseases.

That very similar attacks, if not at times identical, may appear on a purely neurogenic basis is known. Similar pain, but more frequently precordial, occurs in the so-called "effort syndrome." On the other hand, a true angina may readily enough occur in an individual with other symptoms classified as neurotic, and it is really better to assume that it is a real angina or else demonstrate that it is not an angina at all.

There is nothing in the etiology which we can look upon as definite. In general, it is a disease of later life, from the fifth decade on. Apparently typical cases have been reported in children, but it is probable that such cases are referable to some

definite pathology, as acute pericarditis, adhesive pericarditis, or aortitis. Vaquez in his text reports a fatal case in a boy of eighteen, when an aortitis was found at autopsy.

Males are much more frequently affected than females, but in a less proportion than given in the earlier writers. Considered as a whole, it bears no relation to occupation or to lack of occupation, although an undue number of doctors are included in the data. This Osler thought due to the stress and worry of medical practice.

Heredity appears to be some factor, the best known illustration of which is the death by angina of Matthew Arnold, of his father, the "great and good" Thomas Arnold, and, again, of his father.

High blood-pressure has been given as a factor, but the attacks occur quite as frequently with a low or a normal pressure. It is not rare to see attacks first begin when the pressure falls gradually in a patient previously carrying a high pressure and where there is no evidence of a coronary accident to account for the fall in pressure.

Allbutt speaks of the etiologic importance of infection. Certainly syphilis plays a large part, as does aortitis of any origin.

Rheumatic carditis and the resulting valvular lesions are definitely not predisposing factors. Cases do occur, but relatively rarely.

As there is no definite etiology, so there is no definite pathology, unless one chooses to follow those who would predicate aortitis or coronary disease as essential to the diagnosis. Cases which are quite typical clinically show a very varied pathology at autopsy, and cases of angina with death in an anginal attack are reported where there was no demonstrable pathology at autopsy.

Allbutt, and with him a great many others, hold that aortic disease is the underlying factor. Others subscribe to this, but say that in the absence of aortic pathology tension at the base of the aorta, transmitting painful impulses through the depressor nerve, causes the pain.

Whatever view we may hold, we are forced to agree in part,

and say that a great many cases are due to aortic disease. The
proportion may be very great, depending upon the nature of
the material, but that it is the sole and only cause does not seem
to be borne out by autopsy material or by clinical observation.
Many show no changes in the aorta or only such as are a normal
incidence for the age. Intra-aortic tension as a factor meets with
the objection that in a large proportion of cases the blood-pressure
as measured peripherally is normal or lower than normal. To
say that such cases are not true angina means attempting an
artificial clinical classification which cannot be followed by an
actual clinical differentiation at the bedside.

Coronary disease has been held to be the underlying factor
from the time of Jenner. That coronary disease can cause an-
ginal pain we are sure from our cases of coronary embolism and
infarction, but that coronary disease is *the* chief factor is again
not borne out by autopsy material. Some patients who have
died during an attack show no changes or but slight changes,
and others with extreme changes and even occlusion have given
no history of angina, and have not died in an anginal attack.
Angina usually occurs at an age when coronary changes would
be expected, and the proportion is quite the same in those with
and without angina.

It would seem that conditions would best be met by adopt-
ing a more eclectic view.

We know that pain does arise from the viscera and that
when pain does originate in a viscus, it does so by involvement
of the sensory nerves of the same embryologic segment, so that
pain is not referred to the viscus, but to the area ruffled by the
sensory nerves of that segment. As Mackenzie states, there is
but one process by which pain can be produced in a hollow vis-
cus, and that is by contraction of muscle, and contraction of
muscle under certain conditions, as (1) when the contractions are
very strong, (2) when the muscle is not supplied with sufficient
blood; (3) when certain parts of the central nervous system (or
autonomic) are hypersensitive.

Thus we might have pain originating in the heart muscle if
that muscle were called upon to do work, or too much work,

with a deficient blood-supply. This could readily be due to anatomic changes in the coronaries. We would still have to account for those with slight changes or with normal coronaries. If we assume, and we are assuming, as Von Neusser and many others have assumed, that there is an actual vasoconstriction of the coronaries, it will help to explain such cases and to explain many other phenomena in connection with angina.

That such an active vasoconstriction does occur would seem probable, as a nerve supply to the coronaries has been shown by Gordon Wilson, and such a nerve supply is confirmed, at least in part, by experimental physiology. Maas showed that the vasoconstriction fibers from the coronary artery terminated in the vagus. Vasoconstriction and vasodilatation have been demonstrated, but it has been argued that there are passive changes, and objection which has not been completely met. The purin-base diuretics increase the coronary flow, but here again it has been objected that it is due to other causes than active vasodilatation.

Active vasomotor changes would help us to explain much else. In the absence of other factors it would help to explain the age incidence, for we have some reason for thinking that the autonomic system is overlabile with age. It would help to explain the pain very similar to and at times identical with angina in the so-called "effort syndrome," where again we have an autonomic system responding to smaller stimuli, and over-responding when it does respond. It would help to explain the influence of gastro-intestinal factors, of worry, emotion, and fatigue.

In regard to the prognosis, we at least know this very definitely, that we do not know and have no way of knowing, as far as basing any prognosis on the attacks themselves are concerned. The patient may die in the first attack, the second or third, or may survive for years.

In general, the prognosis depends especially upon the underlying pathology, where such pathology can be demonstrated.

A great many minor factors do give some idea of the prognosis, but are too indefinite and uncertain to discuss here. In-

creasing frequency of attacks and the occurrence of attacks during the night are of bad omen. A falling blood-pressure is of serious import here as elsewhere.

The long attacks, lasting hours, and not relieved by the usual methods, are almost invariably more than an angina. Very frequently they are due to added coronary disease. Especially is this true when associated with a fall in blood-pressure and the presence of a ventricular type of rhythm makes it almost certain. The prognosis in such long attacks is, of course, much worse, both as to the immediate attack and the ultimate outcome.

Aortic disease will also produce such prolonged attacks, but offers more in the way of physical findings.

In the management of angina cases I would like to urge one thing at the start, and that is a spirit of common sense optimism. It will add a great deal to their comfort, even if you are mendacious. Their families should, of course, be advised as to the true state of affairs. I can see no reason for being unduly dismal about angina. We have no way of estimating the prognosis, and it would make no difference if we did. It is just as well to assume the more favorable outlook.

I can also see no reason why the ordinary case should not go about his business as usual, with, of course, some very moderate, but quite necessary instructions. Except in some obvious cases you have no reason for thinking that he will die any sooner in his office or on the golf course.

There are certain general rules which should be adhered to. His sudden disability should not be allowed to jeopardize others, or himself, as it would were he allowed to drive a car or run a locomotive. He should be advised not to exert to the point of bringing on the pain and should stop when the pain does come on.

Because of the greater frequency of attacks on a full stomach, and of fatal attacks, he should rest quiet after meals. And he will do better if advised to rest half an hour or more before his meals. The diet should be such as not to produce gas.

The bowels should be kept open without cathartics if possible. I believe the attacks will be less likely to occur without cath-

artics. Three or four glasses of hot water on arising will often
work wonders with the bowels. Plain white vaselin will usu-
ally work well, and is more efficient than the lighter oils and does
not have certain of their disadvantages. It can be kept cold
and a large bolus will slip down easily. If laxatives are neces-
sary, the milder saline laxatives should be used.

Dilute hydrochloric acid after meals may decrease the gas,
or large doses of charcoal night and morning.

For the attacks, nitroglycerin, gr. 1/100, under the tongue
will usually relieve at once. It is more convenient than amyl
nitrite, less unpleasant, and, as far as I have observed, works as
well or better. For the longer attacks morphin may be neces-
sary. Occasionally milder drugs will help, as aspirin, allonal,
and others.

Between the attacks iodids are frequently advised. I have
myself never seen any benefit that I was at all sure of, except
in a few of the frankly luetic cases. They should not be used
to the point of upsetting the stomach or throwing any additional
load on the kidneys. Their use is quite orthodox, but I doubt
their value.

I have already mentioned the effect of the purin-base di-
uretics on the coronary flow. Clinically they are of value more
times than not. Experimentally Heathcote, Sassa, and others
have shown that they do actually increase the coronary flow.
Experimentally they are of value in this order: Euphyllin,
theobromin and its salts, theocin and its salts, and caffein and
its salts. Our own clinical experience has been best with theo-
bromin, either the sodium salicylate or the sodium acetate,
usually in 10-grain capsules, four times a day for three or four
days. If taken continuously it will almost invariably cause such
symptoms as nausea or headache; it also loses its effect. We
have usually given it for only three or four days of each week,
being very careful to avoid ill effects. If ill effects are once ob-
tained, the patient is not very apt to be co-operative when its
resumption is suggested. It is usually taken best before or with
the meals. A few patients tolerate it better after meals. Some
patients cannot use it at all. Theoharri has shown that in his

cases theocin reduced the blood-pressure. In some that we have seen it has, although our results were not as good or as permanent. In high pressure cases it may be the drug of choice. Its effect on the angina is about as favorable as that of theobromin. Euphyllin is not supposed to nauseate, but occasionally it does. It is much better tolerated, however, and has at least almost equal value to theobromin. Caffein is worth trying if the others cannot be tolerated, and its central effects may not be as marked or feared.

Surgery will be of value in selected cases. It is too wide a subject to be discussed here.